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AMERICAN
JOURNAL OF PSYCHIATRY

CONVULSIVE SEIZURES, THEIR PRODUCTION AND CONTROL.

WITH ESPECIAL REFERENCE TO THE PROBABLE MECHANISM OF THE SEIZURE ITSELF.*

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A brief epitome of our present views regarding the acute and chronic manifestations of the convulsive state as a whole are here set forth in terms of a common denominator which may be found so frequently present throughout the symptom complex, as to command serious consideration.

The appearance of convulsive seizures are associated with widely separated diseases and conditions embracing every field of medicine. The acute and transitory phenomena of convulsive seizures noted in the exanthemata of childhood as well as eclampsia, uremia, cerebral trauma and pressure are as important in this consideration as are the chronic forms of the so-called "idiopathic," "symptomatic" and Jacksonian epilepsy.

The symptom complex in its acute manifestations, especially in the eclamptic and uremic, is of particular interest to the practitioner as it claims a large annual toll of life. Even greater is the tremendous loss to the state and community from the chronic and recurrent phase of this so-called disease, which commands the attention of the neurologist and psychiatrist. The most serious manifestation is the major convulsive seizure which is of greatest

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concern as it endangers the patient's life or completely incapacitates him from further economic usefulness.

The milder manifestations, such as twitchings, Jacksonian attacks without loss of consciousness, momentary confusion, aphasia, and transitory disturbances of thought processes, can occur without interference with the individual's general activities.

A careful search for a common denominator which might be associated with the major type of convulsive seizures has disclosed in our opinion a *predisposing* underlying factor which can be isolated and established. It has been possible during the past three years to remove or reinstate the major convulsive seizure almost at will in over one hundred cases, with such certainty that there seems to be little doubt that we are dealing with an inherent motor phenomena subject to variations of cortical control rather than a "disease" of the motor areas.

Sufficient time has elapsed to state definitely that body fluid imbalance and its consequent hydraulic cerebrospinal fluid pressure affects directly or indirectly the frequency and severity of the major attacks and that the post-convulsive manifestations such as headache, vomiting, mental torpor and stupor are due to cerebral edema and intracranial pressure.

During the past two years sufficient confirmation of this theory, both clinical and experimental, has been added by other investigators to justify the belief that a rational means of control of the major seizures is now possible through continued and careful balance of water metabolism of the body.

S. A. K. Wilson,[20] Foster Kennedy [18, 19] and others consider epilepsy as a symptom complex, not a disease. If we go one step further and assume that a convulsion is a *normal* mass reaction of the cortical motor areas and integrated levels, temporarily devoid of inhibitory control and is released to reflex activity by some intercurrent mechanism we will find a plausible and interesting approach to the entire subject.

If we consider the convulsion *per se* in the same light as we do the tendon reflex we would expect the massive response to a sensory stimulus rather than the reaction to a single muscle group. Cortical levels would give a predominance of flexor patterns with extensor inhibiting manifestations and certain refractory periods and repetition according to Sherrington's law. Tendon reflexes have been

considered "normal" because of the ease with which they are elicited and their common acceptance by the profession at large. It is doubtful as to when the human race became conscious of this reaction.

Although reflexes may be obtained in the lower forms of life, it is questionable as to whether the animal is conscious of such a phenomena, and whether it would be considered normal were it recognized. Many human beings are unconscious of the fact that they possess such a reaction and we have all noted the surprise on the part of the patient in certain cases when the patellar reflexes were elicited. A similar reaction might well be expected in the case of a more complicated mass response such as a cortical reflex, involving many muscles and more commonly termed a convulsion. The work of Rowntree,[20] Kubie,[20, 21] Weed [35, 36] and Drabkin [2] indicate that convulsive seizures may easily be produced in the lower animals by the ingestion of large quantities of fluid. Such a convulsive manifestation can also be obtained in man under similar circumstances.

Every normal individual has the potentiality of this reaction (Lennox and Cobb [22]) and under proper preparation of the motor levels to be discharged, might be expected to produce such a phenomena in the same way that a tendon reflex may be produced when proper stimuli are applied at proper points. Since a tendon reflex is dependent upon three fundamental neurological components in its production (*i. e.*, sensory conductors, integrated distributors of this impulse with inhibitory and threshold controls, and finally motor cell expression to the muscle) it is along these three sides of the arc that methods of interruption must be attempted if we are to check or control the major responses from the cortical areas, designated as convulsive seizures.

This assumption therefore implies that the problem of violent mass motor responses (convulsions) might be successfully attacked along three lines:

(1) By diminishing or preventing the sensory impulse responsible for the motor (mass) reaction.

(2) By raising the inhibitory threshold and interrelated controlling centers to prevent such stimuli from spreading throughout highly integrated cortical and subcortical levels, and thus checking or localizing such mass reactions.

(3) By depressing or destroying the motor elements responsible for the externalization of the discharge.

It is evident that the latter method is undesirable. Successful therapy has rotated around the two former considerations. It is also evident that in this concept, little can be expected from attempts to place the lesion responsible for the attack *within* the nervous system, as what is an inherent quality of a motor level and its response in a normal manner, cannot contain a primary epileptogenic pathology, neither can a diseased or depressed motor cell be expected to function with the violence and duration that characterizes a generalized convulsion. The normal state of activity which so promptly ensues following a major attack and the absence of pathological motor signs between attacks strongly support this view (late and terminal states as well as organic lesions considered elsewhere (Fay)). The futile search for a characteristic pathology might thus be expected.

In the light of our interpretation, epilepsy and the convulsive state must be divided into (a) *minor,* transient, focal manifestations without loss of consciousness (which will not be discussed in this paper), and (b) *major,* generalized convulsive seizures with loss of consciousness and varying post-convulsive sequela. The major seizures must be considered in the light of an intermittent, variable, fulminating, explosive response of the motor areas, the patient free from such manifestations and symptoms at one time and subject to violent neurological disturbances at another.

The major seizure has commanded our attention and its addition and subtraction from the minor state has been clinically possible during the past three years. A search for the variable which determines the occurrence of major attacks and permits periods of relief led in 1927 to the consideration of fluid and water metabolism as the factor responsible for the periodicity.

Gamble [12] has recently shown a characteristic tendency on the part of the epileptic toward water storage during the interval between the attacks, with a definite release of this fluid immediately following the seizure. He has further been able to determine that this fluid storage has been maintained in the interstitial compartment which is dependent upon the fixed base of sodium (Na.) for its fluid volume. He points out that the cerebrospinal system

represents one of the largest single reservoirs of interstitial fluid in the body.

Improper elimination of fluid is also closely related to attacks as shown by McQuarrie [24] who demonstrated that if pituitrin were given to an epileptic patient renal elimination was diminished and the attacks precipitated, increased in number.

Fremont-Smith [12] demonstrated the decrease in urine production and the tendency towards hydremia when pituitrin was given to the hydrated experimental animal.

Fluid ingestion and absorption play a large rôle in the rapid acquisition of fluid by the individual, its mobility, storage and elimination depending upon the many factors surrounding water metabolism.

In order that over-accumulation of fluid within the subarachnoid spaces may arise, a deficiency or decompensation in cerebrospinal fluid elimination must be present to account for the tendency which certain individuals manifest toward convulsive seizures and the apparent freedom experienced by the majority of human beings. The factors surrounding the deficiency in cerebrospinal fluid elimination must be variable within a wide limit, so that certain cases would show almost complete compensation for overloads or retention of fluid and other cases represent the poorest type of compensation for this fluid variable.

The close relationship between fluid ingested and the prompt production of cerebrospinal fluid has been well established by Weed,[37] Kubie,[20] Howe [16] and many others. In order that over-accumulation of cerebrospinal fluid may occur with its secondary intracranial pressure disturbances, a search for pathological involvement of the outlets of cerebrospinal fluid as well as obstruction in the pathways has been sought. A definite pathological involvement of these structures has been demonstrated by us [9, 40, 41] and deficiencies in the venous drainage system favoring passive congestion noted by Swift.[32]

Thus the factors surrounding the increase of subarachnoid spinal fluid and the preparation of the patient for an intermittent or chronic intracranial pressure has been established. The well known fact that the convulsive state whether acute or chronic is associated with increased amounts of supracortical fluid has been recognized since the days of Hippocrates [15] and definitely established in patients

suffering from this condition by means of encephalography (Pancoast and Fay [27]) as well as many direct observations of the cortex with the brain exposed at operation. The demonstration of a chronic slightly increased intracranial pressure by Patterson and Levi [28] and in our own series, as well as the familiar finding of increased pressure at the time of a convulsive seizure adds important confirmation to these views.

If we now consider this increased fluid from the standpoint of hydraulics within an almost closed cranial cavity, it is possible to demonstrate that convulsive seizures occur in the clinical groups, where this fluid increase occurs in the frontoparietal areas of the brain, and thus may be considered as specifically applied intracranial pressure.

As the presence of cerebrospinal fluid and its mobility is mostly concerned with water metabolism and hydraulic distribution within the closed confines of the skull we must seek the expression of this pressure in terms of fluid accumulation and elimination not only from the cerebrospinal fluid system, but the body itself. The sequence of physiological events, secondary to this pressure, and promoting its sudden activity, are almost too numerous to detail. Venous obstruction, hypertension, hydrated states, renal and cardiac insufficiency, anoxemia, alkalosis and carbohydrate metabolism are intimately concerned with the accumulations of tissue fluids. Exertion, straining and cerebral vasomotor disturbances register their immediate effects upon cerebrospinal fluid pressure. In the presence of over-accumulations of fluid these responses may be greatly exaggerated and produce abnormal degrees of pressure.

We have established, we believe, that this specifically applied cortical hydraulic pressure is responsible, either primarily or secondarily, for release of the controlling inhibitory centers, which permits a generalized motor mass response to an appropriate stimulus. The character, source or origin of the sensory stimulus may be variable and need not concern us more than would the same factors concerned in a simple tendon reflex. Of most importance is the fact that a *major* convulsive seizure is possible only when general increase in subarachnoid fluid is present, under specifically applied conditions. I [10] have pointed out that the major convulsive seizure including the stupor, vomiting and headache can be entirely eliminated from the cycle of events by control of the cerebrospinal fluid

volume and pressure. This leaves Jacksonian epilepsy, petit mal, with the many variations known as subcortical epilepsy, etc., as imperfect or partially inhibited motor responses, and as might be expected, capable of major manifestations dependent upon increasing application of the pressure, or variable as to the remaining cortical areas of control.

Clinically we have shown that absolute control and balance of fluid intake, and thus secondarily of cerebrospinal fluid accumulation, during the past three years has relieved and eradicated the grand mal seizures in those cases where a true fluid balance has been obtained. A few severe chronic cases have been encountered where the control of fluids, to a sufficient degree seemed incompatible with life, because of profound organic disturbances and hence have been without improvement on the part of the patient.

The symptom complex thus disclosed has seemed to us to be analogous to that of diabetes, where a sugar free state may be obtained by proper adherence to diet, admitting a few cases where this becomes impossible without the aid of insulin because of the severity of the condition. A sugar free state may usually be maintained as long as strict balances are obtained. Immediate return of sugar occurs with indiscretions of the diet. There, of course, has been no cure for the basic pathology of this symptom complex, the tendency to a diabetic syndrome remains. The same may be said for chronic major epilepsy, a *control* of the seizure is possible on a strict fluid balance, but return of attacks may be expected when indiscretions in fluid and diet occur.

The series of epileptics studied includes all varieties, early and late—" idiopathic," " symptomatic," " posttraumatic," " Jacksonian " and " psychic-equivalent."

Occasionally six months to one year has been required to establish a proper balance of fluid intake and output and control in the long-standing institutional type of patient, because of mental deficiency and lack of close cooperation. However the usual period required to obtain a satisfactory balance in the cooperative patient varies between three to six weeks.

Where mental deficiency has become well established, we have found the release of the patient from the major seizure gives rise frequently to a " behavior " problem, which is even more undesirable and difficult to manage than the condition formerly presented

by frequent convulsive seizures, prior to dehydration. It is our distinct feeling, therefore, that those cases with mental deficiency are hopeless from the standpoint of economic readjustment of the patient. Preventive and progressive measures should be directed toward the early cases so that the hydraulic factors concerned with the atrophy of the brain (Fay,[8] Winkelman and Fay [41]) may save the patient from being institutionalized in the future, as well as permitting, by control of the major seizures, his proper mental and social development to some form of economic usefulness.

It is my considered opinion [3, 4, 5, 6, 7, 8, 9, 10, 21] that the repeated, acute, intracranial pressure waves, during and following a major convulsion, are responsible for prolonged periods of supracortical pressure causing low-grade cerebral anemia and ischemia, with consequent atrophy and the characteristic dropping out and degeneration of the ganglion cells, in specific areas of the brain exposed to the influence of this *"hydraulic cast"* so perfectly applied to the frontoparietal areas of the cortex which lie within the cerebrospinal fluid circulating field.

This secondary degeneration is probably responsible for the progressive mental deterioration. The hope for future progress in this problem therefore lies in the early prevention of this fluid pressure, as we believe the cause to be chronic hydraulic pressure.

Discussion.

According to S. A. Kinnear Wilson [39] heredity can only account for 16 per cent to 20 per cent of all cases. In this group, structural deficiencies of the cerebrospinal fluid eliminating mechanism, such as the Pacchionian bodies (Winkelman and Fay [41]) and venous drainage channels; especially sigmoid, lateral and occipital sinuses (Swift[22, 23, 24]) where great anomalies occur in the idiopathic cases, may prove to be the "hereditary factor" in this group. Such aplasias and anomalies we believe give rise to serious disturbances of cerebrospinal fluid elimination and consequently poor compensation for periods of spinal fluid overload.

We [10, 41] believe the remaining 80 per cent of cases rests upon damage to the subarachnoid cortical fluid pathways or the filter structures, (a) the *Pacchionian bodies* at the vertex, or (b) *disturbance in venous return* from the brain, including lesions situated

between the skull and the heart, acquired during fetal life or after birth. Birth trauma, subsequent cerebral injury with bloody spinal fluid, inflammation, tumors and infections, are factors consonant with the former. Impairment in jugular drainage due to mastoid inflammation, tonsillar abscesses, growths in the neck, enlarged thymus, patulous ventricular septum of the heart and Stokes-Adams' manifestations are frequently found in the latter. As no valves exist in the jugular veins, obstructive lesions or high intrathoracic and venous pressures register immediately upon the enclosed cerebrospinal fluid field and further enhance the pressure by the effects of edema and passive congestion.

The whole question of fluid elimination and retention in the body becomes most important in this light. The kidney function, and its variability; skin elimination and its deficiency under hyperpyrexia; the entire question regarding fluid intake, its rapidity of absorption into the blood compartment, and its rapidity of distribution into the interstitial compartment (of which the cerebrospinal fluid represents the largest single reeservoir (Gamble [13])); the factors surrounding water storage; and glucose metabolism of the tissues are all registered in terms of cerebrospinal fluid production and over-accumulation and must be evaluated in terms of deficient filter and drainage mechanisms and the consequent effects in each case. Uremia, eclampsia, and the acute infections of childhood frequently are associated with convulsive seizures and almost invariably with a hydrated state. The forcing of fluids on the patient by the practitioner to " combat the toxemia " usually greatly enhances the disturbed water metabolism and storage rapidly inducing the factors most favorable to the production of a convulsive seizure.

Water storage in the presence of carbohydrate metabolism, and the greater need of the cell for fluid, under this type of metabolism (Gamble), as well as the relationships of organic and inorganic substances making up the " fixed base " and consequently fluid volume of the interstitial and blood compartments thus disturbing the water balance and its mobility, may we believe, determine the periodicity of attacks.

The periodic storage of fluid is seen in the normal, as well as in the epileptic (Fay,[6] Gamble,[14] McQuarrie [25]) and becomes a most

important factor where deficiencies in compensating for this over-accumulation may lead to the advent of the convulsive cycle of events.

Behind carbohydrate metabolism lies insulin and the attacks of hypoglycemia (Drabkin and Shilkret,[1] Ravdin,[29] Howland,[17] Nielson[26]) and the probable secondary interrelation of the adrenal, liver, anterior pituitary (Wilder[35]) as yet undetermined. The group of epileptics suggesting glandular types may well be considered from this standpoint.

The physiological determinations surrounding an attack have been carefully considered by many observers (Lennox and Cobb[22]). Anoxemia, the effect of slight changes of the PH to the acid side on the oxygen dissociation curve, CO_2 relationships, alkalosis, edema and increased permeability, all influence capillary activity to fluid.

The cycle of events surrounding a convulsion might be summarized as follows:

(1) Unrestricted intake of fluid.

(2) A disturbed threshold for fluid elimination with storage in the "intestitial compartment."

(3) Interstitial water storage (carbohydrate metabolism, alkalosis, increased fixed base (Na.), venous stasis).

(4) Improper fluid elimination (kidney and skin).

(5) Increase in subarachnoid volume and pressure (hydraulic (spinal fluid) pressure).

(6) Hydraulic pressure exerted on the frontoparietal areas of the brain in the supracortical cerebrospinal fluid pathways.

(7) Gradual capillary insufficiency with increasing anoxemia, due to increased ratio of fluid to blood within the closed confines of the skull.

(8) Increased permeability of tissue (secondary to anoxemia).

(9) Tissue edema (perivascular, subcortical).

(10) Limits of physiological compensation reached for variations.

(11) A proper sensory stimulus (visceral or parietal, central (including subminimal cortical irritation by traction from arachnoid adhesions, Lind); or peripheral).

(12) Vagotonic phase (emotion, hypoglycemia).

(13) A sudden cerebral vasomotor response with fall in general systemic blood pressure producing almost complete cerebral anoxemia.

(14) Sudden rapid increase in intracranial pressure (Forbes and Wolff [11]), tissue edema.

(15) Sharp application of hydraulic supracortical pressure on the exposed gray matter of the motor cortex with releasing of higher inhibitory centers.

(16) Decerebration with syncope and loss of predominant flexor patterns of the cortex.

(17) The phase of decerebration, *tonic spasm.*

(18) Rise in systematic blood pressure (intrathoracic pressure and spasm of the large muscles) and adjustments of cerebral cortical circulation.

(19) Return of circulation to the cortex, sufficient to permit physiological activity of the motor cells (oxygen, CO_2 exchange). (Sufficient circulation must occur to give proper functional exchanges otherwise paralysis similar to that seen in thrombosis would follow. A dead or imperfect cell cannot produce violent reactions.)

(20) Reflex mass action or "discharge" of the cortical or released integrated motor level.

(21) Shift of the PH to the acid side (increased CO_2 from apnœa and lactic acid from muscles).

(22) Disappearance of cyanosis with return of respirations.

(23) Better utilization of oxygen under PH shift to the acid side with lowering of the oxygen dissociation curve.

(24) Reversal of tissue permeability and passage of fluid from the interstitial compartment to the blood vascular compartment.

(25) Final exhaustion of the motor area from activity or increasing intracranial pressure.

(26) Post-epileptic continuation of pressure and cerebral edema, maintaining physiological interruption of consciousness with stupor and sleep.

(27) Elimination of large quantities of fluid (skin and urine) and readjustment of vascular and cerebrospinal fluid values.

(28) The return of the patient to a "normal" condition.

(29) The variable interval between attacks, during which there may be a gradual re-establishment of over storage of fluids and preparation for another convulsant cycle.

NOTE.—Status epilepticus, epilepsia partialis continua, and certain numerous and persistent Jacksonian attacks, in my experience, have all been associated with collections of *extraarachnoid* fluid, tumor, or adhesions proved by operation, not capable of being absorbed or removed, except by direct operative means. In every other type, either by encephalogram or operation, or both, increase in the subarachnoid fluid has been found, whether in eclampsia without previous history of convulsions, or in the so-called chronic idiopathic epilepsy.

The hope of controlling these major attacks lies in establishing a balance and threshold for fluids, within the individual capacity of the patient to properly handle the amount of cerebrospinal fluid produced and absorbed or with due respect to the defective vascular drainage or body eliminating mechanism. As spinal fluid volume as well as interstitial fluid volume depends upon the fixed base, carbohydrate and insulin metabolism and other factors surrounding water metabolism, the better understanding of this phase of the problem must await investigation in this field. Contributing factors such as increased cerebral vascular circulation, deficient venous drainage, maintenance of proper oxygen supply at all times, renal function, cardiac function, blood pressure and gastrointestinal problems concerned with absorption of fluid and excretion of fixed base become closely associated with the physiology of water metabolism.

The method so far evolved, that has given the greatest amount of attack free results, is the strict limitation of total fluid intake to below 20 ounces per day, a solid " dry " diet sufficient to meet the needs of the skin, breath and bowels without permitting the demonstrable increase in urinary output. A careful balance of intake and output to within one to three ounces per day must be established by proper regulation of fluids and diet.

Hydration states due to whatever pathology or disturbance in fixed base and vascular and tissue fluid relations should be avoided and adjusted if possible. A search for all mechanical circulatory deficiencies, as well as the factors surrounding proper elimination and especially those concerned with carbohydrate metabolism and water storage must be determined. In every case where an organic

lesion has not been demonstrable disappearance or the control of the grand mal seizures has occurred when proper balances were obtained.

With the addition of fluid beyond the point of compensation, major seizures have promptly returned. Thus dietary and fluid indiscretions as well as intercurrent disturbances in elimination and circulatory function have been found responsible for occasional convulsive episodes which may interrupt the attack free state under dehydration and fluid control.

Where acute generalized convulsive seizures have appeared in diseases of childhood, eclampsia, and uremia, hydration states have been found in every instance, due to either the forcing of fluids beyond the patients compensation or possibilities of elimination, or disturbance in the factors which determine water storage and elimination.

Where an acute hydration state is present, dehydration must be carried to its logical establishment by means of spinal drainage, intravenous hypertonic glucose solution, purgation with hypertonic saline and strict fluid limitation. Only the fluid necessary to maintain circulatory and body functions should be permitted if control of prolonged and severe generalized convulsions is to be obtained.

The direct relationship between fluid intake, its storage and elimination and major convulsive seizures we believe has been clinically and experimentally established. The secondary factors surrounding cerebrospinal fluid, pressure specifically applied over the exposed gray matter of the motor cortex (frontoparietal area), hydraulic pressure followed by mass cortical releases have been discussed and analyzed elsewhere.[10] A rational means of definite fluid balance has been established. The results have justified the conclusions that the major form of the convulsive state is a superadded phenomena associated with hydration and disturbed water metabolism.

We have come to regard convulsive seizures as representing a normal mass reaction of the motor areas after inhibitory centers have been depressed or released by specifically applied pressure, anoxemia or exhaustion. In the series of cases studied the grand mal attacks have gradually receded into Jacksonian or petit mal manifestations as dehydration was accomplished. The petit mal

seizures have increased in severity and the zone of response widened as fluids and intracranial pressure have been increased.

We have therefore felt justified in assuming that the stimulus capable of producing a petit mal or Jacksonian attack at one time finds a wide field of application and permits a generalized cortical release when inhibitory factors have been removed by pressure secondary to hydrated states.

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SOME OBSERVATIONS ON EXPERIMENTALLY PRODUCED CONVULSIONS.

II. The Type of Convulsions Elicitable After Lesions of the Rubro-spinal System, with Some Incidental Findings.*

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I. General Statement of the Problem.
   1. The basis for the view of shock and inhibition versus the view of the change in the functional capacity of a conduction pathway.
   2. The method of successive lesions as a means of attack upon the problems of organization of the motor and sensory systems.
   3. The graphic representation of the number of possible combinations of lesions, together with the particular effects of any given pair.

II. The Experimental Results.
   1. Lesions of the pyramidal system.
      A. Ablation of the cortical motor areas.
      B. Median incision of the pyramidal decussation.
      C. Ablation of the cortical motor areas followed after an interval by median longitudinal incision of the pyramidal decussation.
      D. Median incision of the pyramidal decussation followed after an interval by cortical motor area ablation.
   2. Lesions of the rubro-spinal system.
      Median incision of the midbrain and severing the decussation of Forel.
   3. Combined lesions of the rubro-spinal and pyramidal systems.
      A. Median incision of the midbrain followed after an interval by median incision of the pyramidal decussation.
      B. Median incision of the midbrain and ablation of the cortical motor areas.
      C. Median incision of the midbrain, incision of the pyramidal decussation and ablation of motor areas.
   4. Combined lesions of the rubro-spinal and vestibular systems.

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5. Obstruction to the venous outflow of the brain.
   A. Ligation of the external and internal jugulars of both sides and of one vertebral vein.
   B. Blocking the transverse sinuses.

A year ago we [1] presented for your consideration some results on the localization of the cells of origin for the nervous impulses leading to the tonic and clonic muscular manifestations in experimentally induced convulsions. At that time we laid some emphasis upon the method of successive lesions within the central nervous system as a means of investigating the general neurological foundation of some of our present day beliefs in the field of the functional organization of the central nervous system. We called attention, also, to the view of Hughlings Jackson [2] that a change in the quantity of nervous energy passing through the remaining pathways or levels or systems of the central nervous system after an injury to one of them might account for the recovery, always more or less incomplete,[3] from any such injury compatible with the maintenance of life. The method of successive lesions has yielded results which, in our opinion, are more easily and logically explained on the basis of Jackson's idea than on the more commonly accepted idea of inhibition and shock as developed by Setschenow [4] and by Goltz [5] and his school. We emphasized, also, the view that, during the phylogenetic development of vertebrates in the course of organic evolution, there has been a wandering of function from the lower, phylogenetically older mechanisms to the higher, phylogenetically newer mechanisms of the cerebral hemispheres, so that it is obviously incorrect to conceive of the same level or division as having the same functions in all types of vertebrates. Since there is some confusion in the minds of many as to the actual basis for the view that the spinal cord has the same reflex and other functions in man that it has in the frog, or that the cerebral hemispheres have the same functions in the two forms, we may refer to Goltz's original statement.[6] In order to support this view, Goltz found it necessary to make another assumption—the assumption of shock. His statemay be given in Loeb's [7] translation:

> No one will assume that that piece of the spinal cord which is separated from the brain in so short a time (*i. e.*, a few days or weeks) acquires entirely new powers as a reflex organ; we must assume that these powers were only temporarily suppressed or inhibited by the lesion of the spinal cord.

Goltz extended this line of argument to show that the motor cortex of man or of the dog could not be an important part of the motor mechanism, since there is a recovery of function after an interval. The only reason, as Goltz argued, that man did not recover as completely as the frog was that the shock in man was greater.

Goltz's view of the suppression of function by inhibition or shock, and Hughlings Jackson's view of the change in the quantity of nervous energy flowing through the remaining pathways after a lesion of one or more structures, come into sharp conflict. Goltz takes no account of the changes in structure during phylogenetic development, whereas, Jackson's view is a necessary supplement to the view of the wandering of function toward the anterior end of the central nervous system during vertebrate evolution. For, unless some change in the capacity of a conduction pathway after injury to some other path can be shown, it would be difficult to show that the cerebral hemispheres, for example, do not have the same functions in the frog as in man.

We wish to add to our former statement, certain new experimental results, obtained by the method of successive lesions, which seem to us to indicate even more clearly the essential participation of the pyramidal system in the clonic manifestations of epileptic convulsions when this system is intact, and to show even more clearly than we had done the dependence of the tonic manifestations upon the lower motor mechanisms, particularly the rubrospinal system.

The method of successive or combined lesions has been used by others,[8] but it does not appear to have been used extensively or intensively with the particular idea in mind of analyzing the mechanism of compensation for injury. In one respect, it presents an almost bewildering complexity which might, at first sight, seem to preclude an adequate analysis of the function of the nervous system during the lifetime of any one investigator. If the names of the various structures of the nervous system are arranged in a vertical column at one side of a diagram, say the left, and the same names in the same order are then arranged in a horizontal line at the top or bottom of the diagram, one has the familiar form of chart in which one property or activity—*e. g.*, pulse rate or respiratory rate—may be plotted against any other property or activity, as body

temperature. In our diagram, however, the various parts of the nervous system are plotted against each other. If one hundred different structures or parts of the nervous system are taken to start with (and it requires no great degree of anatomical refinement to get at least one hundred different structures in the nervous system), it is evident that about ten thousand different combinations of two lesions each are possible. A lesion of the motor cortex cerebri, for example, may be combined with a lesion of any one of the other ninety and nine structures in the list. In some cases, it will be found, on experiment, that a lesion of a second structure, inflicted at some interval after the primary lesion of the cerebral cortex, will considerably increase the gravity of the symptoms which result from the lesion of the motor cortex alone, while others may be almost or quite without effect upon the motor symptoms. Deafness, resulting from an injury to the auditory portion of the ear (without involvement of the vestibular or non-auditory portion) does not increase the motor symptoms seen in hemiplegia. Whereas, one might imagine that a lesion of the red nucleus might have more severe effects, when inflicted after an interval of recovery from a previous lesion of the motor cortex, than when a lesion of the red nucleus alone is made. It will be apparent from these considerations that the conquest of the functional organization of the motor system is scarcely to be made by a single brilliant cavalry charge or by a single infantry assault upon the enemy's line, but that the more gruelling and less spectacular methods of trench warfare must be adopted. For our purpose, it is not necessary to make attacks upon the whole line of entrenchments, but to pick out those sections which seem most vulnerable to attack, or which would seem likely to yield results of most significance to the immediate problem of the functional organization of the motor system.

These considerations may be brought out more clearly by reproducing the list of structures in the nervous system of which it seems desirable to get localized or circumscribed lesions, first alone or singly, and then in combination with some other structure or structures. The structures of the afferent system are given first, beginning at the periphery and proceeding toward the cerebral cortex. The structures of the efferent system follow, beginning at the cerebral cortex and proceeding toward the periphery. But it should be remembered that, in many of the structures, it is not

possible to separate sharply or completely, the afferent from the efferent components. With our present methods, we must be content with mixed lesions rather than expecting the attainment of the ideal separation into purely afferent or purely efferent. And in the case of many of the structures, we cannot hope always to limit the lesion to one single structure, but must involve some others with it. In the case of a dorsal or ventral root of a spinal nerve, we may be fairly sure of getting only afferent or only efferent fibers, and of getting only the structures which we desire. But in the case of the decussation of the pyramidal tracts or of the decussations of the rubro-spinal tracts, we cannot be so sure that we have involved efferent fibers alone, or that we have limited the lesion to one or the other decussation alone.

THE ORDER OF ARRANGEMENT OF STRUCTURES IN THE CENTRAL NERVOUS SYSTEM.

1. Afferent peripheral nerves.
2. Dorsal roots of the spinal nerves.
3. Substantia gelatinosa and entrant zone.
4. Dorsal funiculi.
5. Dorsal spino-cerebellar tract (Flechsig).
6. Ventral spino-cerebellar tract (Gowers).
7. Spino-thalamic tracts (a) lateral.
8. (b) ventral or medial.
9. Helweg's tract.
10. Gracile and cuneate nuclei.
11. Decussation of the lemniscus.
12. Medial lemniscus.
13. Inferior olive.
14. Corpus restiforme.
15. Cerebellum (a) vermis.
16. (b) lateral lobes.
17. (c) nuclei i. Dentate.
18. ii. Emboliformis.
19. iii. Globosus.
20. iv. Fastigii.
21. Brachium pontis (a) peduncle.
22. (b) decussation (median raphe).
23. Brachium conjunctivum (a) peduncle.
24. (b) decussation.
25. Inferior colliculus.
26. Superior colliculus.

27. Thalamus (a) medial.
28. (b) lateral.
29. (c) peduncles.
30. Thalamopallial tracts.
31. Cerebral cortex.
32. Hypoglossal nerve.
33. Vagus (a) ganglion jugulare.
34. (b) ganglion nodosum.
35. (c) fasciculus solitarius.
36. Glossopharyngeal (a) ganglion jugulare superius.
37. (b) ganglion petrosum.
38. Octavus (i) cochlear (a) ganglion spirale.
39. (b) nucleus accessorius.
40. (c) nucleus olivarius superior.
41. (d) corpus trapezoideum.
42. (e) tuberculum acusticum.
43. (f) lateral lemniscus.
44. (g) corpus geniculatum internum.
45. (h) temporal lobe.
46. (ii) vestibular (a) Scarpa's ganglion.
47. (b) nucleus medialis.
48. (c) nucleus lateralis (Deiter's).
49. (d) nucleus superior (Bechterew).
50. (e) tractus vestibulo-spinalis.
51. Facial; ganglion geniculatum.
52. Trigeminal (a) gasserian ganglion.
53. (b) mesencephalic nucleus.
54. (c) sensory nucleus V.
55. (d) cerebral cortex.
56. Optic (a) retina.
57. (b) optic chiasm.
58. (c) corpus geniculatum internum.
59. (d) pulvinar.
60. (e) optic radiations.
61. (f) occipital cortex.
62. Olfactory (a) bulbus olfactorius.
63. (b) rhinencephalon; uncus.
64. (c) rhinopallium.
65. Motor cortex cerebri.
66. Pyramidal system (i) crossed (a) corona radiata.
67. (b) internal capsule.
68. (c) pyramidal decussation.
69. (d) crossed tract in spinal cord.
70. (ii) direct; lesions in spinal cord.
71. Corpus callosum and association systems.

72. Corpus striatum (i) lenticular nucleus (a) putamen.
73. (b) globus pallidus.
74. (c) claustrum.
75. (ii) caudate nucleus.
76. Pallio-ponto-cerebellar system.
77. Thalamus.
78. Decerebration.
79. Mesencephalon (a) nucleus ruber.
80. (b) decussation of Forel.
81. (c) decussation of Meynert.
82. (d) substantia nigra.
83. Transection at cephalic border of inferior colliculi (a) unilateral.
84. (b) bilateral.
85. Transection at cephalic border of pons (a) unilateral.
86. (b) bilateral.
87. Transection at lower border of pons.
88. Miscellaneous transections of the medulla oblongata.
89. Transection at lower border of medulla.
90. Hemisection of the spinal cord.
91. Transection of the spinal cord.
92. Ventral roots of the spinal nerves.
93. Third cranial nerve.
94. Fourth cranial nerve.
95. Fifth cranial nerve.
96. Sixth cranial nerve.
97. Seventh cranial nerve.
98. Ninth cranial nerve.
99. Vagus.
100. Spinal accessory.
101. Hypoglossal.
102. Cervical sympathetic.
103. Stellate ganglion.
104. Thoracic sympathetic chain.
105. Lumbo-sacral chain.
106. Inferior mesenteric ganglion.

It would indicate a considerable degree of attainment in neurological lore if one could give a clear and unequivocal statement of the exact effects, immediate and remote, of a circumscribed lesion of each of the structures in the list taken singly. If we had this knowledge to begin with, we would have a valuable basis for comparison and control of the effects of a second lesion, added, either immediately or after an interval, to the first. Unfortunately, experimental procedures have not yet covered all the structures in

the central nervous system, and our opinions are often based upon clinical observation of the effects of lesions which frequently are far from being either complete or circumscribed. We are still in that stage of development where the chapters in our text books bear such titles as "The Functions of the Midbrain" with little or no intimation that the midbrain has a relationship to many diverse functional mechanisms which, as yet, we have not sufficiently analyzed. Since we must know the effects of lesions taken singly before we can evaluate the effects of an added lesion, the first procedure is often to determine the effects of a single lesion of each of a series of structures and then make successive lesions of different combinations of two or three of these structures.

But if there is a lack of knowledge of the effects of circumscribed lesions of single structures, there is, a *fortiori*, a much greater lack of knowledge of the effects of combined lesions. We must expect, therefore, to find that the chart showing the effects of combined lesions will consist, at present, largely of blank spaces.

The variation in the effects of a second lesion upon either the immediate symptoms or those persisting after an interval of recovery from the first, may be indicated, for the purposes of the construction of the chart, by the signs +, − and o. If the gravity of the effects due to the first lesion is increased by the second, this is indicated by putting a + sign in the appropriate rectangle on the chart. Warrington states that the chromatolysis in the ventral horn cells following anatomical division of a ventral root of a spinal nerve is increased by homolateral hemisection of the spinal cord above the level of the root which is divided. This effect would be indicated on the chart by putting a + sign in the rectangle in the vertical column under 90 (hemisection of the spinal cord) and in the horizontal line after number 92 (ventral roots of the spinal nerves). The extensor rigidity of the limbs which appears after decerebration is abolished by section of the dorsal roots of a limb, or by section of the dorsal and ventral spinocerebellar tracts. (Sherrington.) In the horizontal line after decerebration—number 78−, a − sign would be placed in the rectangle in the vertical column under number 2 (dorsal roots of the spinal nerves) and in the rectangles under numbers 5 (dorsal spinocerebellar tract) and 6 (ventral spinocerebellar tract). When the second lesion has no effect upon the symptoms due to the first, this is indicated by the

sign 0. The respiratory symptoms seen after bilateral division of the vagi are not affected by division of the 5th cranial nerve proximal to the gasserian ganglion. This would be indicated by putting a 0 under number 52 opposite number 33. A + sign under number 86 opposite number 33 indicates that the respiratory effects of division of the vagi are increased by bilateral transection at the cephalic border of the pons.

The chart does not give detailed information as to the effects of any single lesion or combination of lesions, but merely tells whether certain facts and relationships are now known or unknown. It is a balance sheet, and the details must be sought in the records of the experiments. The balance sheet, when complete, should tell us just where to look in the records for any desired fact or relationship, but the balance sheet cannot be completed until the records are completed.

SECTION OF CHART, SHOWING THE EFFECTS OF COMBINED LESIONS IN THE CENTRAL NERVOUS SYSTEM.

(The original chart was copyrighted 1929 by F. H. Pike.)

No.	Name of structure		1	2	3	4	5	6	7	8	9	10	11
1.	Aff. per nerves.....	..	1	..	..	..	..	..	..	..	..	..	..
2.	D. roots sp. ner.....	..	..	2	..	..	..	..	..	..	..	..	..
3.	Sub. gel. & c.....	..	..	..	3	..	..	..	..	..	..	..	..
4.	Dors. funiculi.	..	..	..	..	4	..	..	..	..	..	..	..
5.	Dors. sp-cereb. tr.....	..	..	..	..	..	5	..	..	..	..	..	..
6.	Vent. sp-cereb. tr.....	..	..	..	..	..	..	6	..	..	..	..	..
7.	Sp.-thal. (a) lat.....	..	..	..	..	..	..	..	7	..	..	..	..
8.	Sp.-thal. (b) vent.....	..	..	..	..	..	..	..	..	8	..	..	..
9.	Helweg's tract.	..	..	..	..	..	..	..	..	..	9	..	..
10.	Grac. & cun. nucl.....	..	..	..	..	..	..	..	..	..	..	10	..
11.	Dec. lemniscus	..	..	..	..	..	..	..	..	..	..	..	11

II. THE EXPERIMENTAL RESULTS.

The technique of the various experimental procedures discussed in this paper has been given elsewhere* and is not repeated here, with the exception of the results on obstruction of the venous outflow from the brain. This is given in a later section.

I. LESIONS OF THE PYRAMIDAL SYSTEM.

By way of recapitulation we may cite the effects of lesions of the pyramidal tract.

A. Ablation of the cortical motor areas. There is severe motor disability as a result, and the type of convulsions elicitable immediately after such a lesion is tonic. There is a remission of the motor symptoms after an interval and clonic convulsions may be obtained thereafter. It was shown many years ago [10] that compensation for the loss of one motor area does not occur through the motor cortex of the opposite side.

B. Median incision of the pyramidal decussation. There is again a very marked motor disability following the injury. The convulsions elicitable immediately afterward are tonic in nature. There is a greater remission of symptoms after an interval of recovery than occurs after bilateral ablation of the cerebral motor cortex, and clonic convulsions are again elicitable.

C. Bilateral ablation of the cortical motor areas followed after an interval by median longitudinal incision of the pyramidal decussation. As is to be expected, the motor disturbances following the second lesion are slight and transient. When the pyramidal decussation is split after an interval of recovery from unilateral ablation of a cortical motor area, the motor effects upon one side of the body are slight, but of the usual severity on the other. This procedure should, theoretically, afford a control of the extent to which incidental injury to commissural neurones in the upper portion of the spinal cord and lower portion of the medulla oblongata complicates the picture seen after median incision of the pyramidal decussation when the cerebral motor cortices are intact. The results so far indicate that any extraneous effect is slight.

One should note here the similarity of this procedure to Sherrington's [11] successive transections of the spinal cord, only the first of which had any effect upon the reflexes, and his conclusion that trauma qua trauma is not the cause of so-called spinal shock should be borne in mind.

D. Median longitudinal incision of the pyramidal decussation followed after an interval by ablation of the cerebral motor cortices. The situation is a bit more complicated than at first sight might appear, and was not considered in our former paper. The

direct pyramidal tract is rather small in the cat and probably does ont descend very far in the spinal cord. We have no information concerning the homolateral pyramidal tract in cats and it is therefore impossible to say whether any uncrossed fibers, other than those of the direct tract, escape injury in median incision of the pyramidal decussation. At first sight, it might be supposed that, since the direct pyramidal tract in cats is not well developed, and since the homolateral fibers, if present at all, are probably not very numerous, the effects of bilateral motor area ablation after an interval of recovery from splitting the pyramidal decussation would be relatively slight. As a matter of fact, the effects of bilateral motor area ablation after an interval of recovery from splitting the pyramidal decussation are severe. It seems wholly improbable that the severity of the effects of the second operation is due solely to the lesion of the remaining pyramidal fibers, or that the effects of the second operation are an accurate index of the function of these remaining fibers.

It should be remembered, however, that, in addition to possible collaterals, given off to the corpora striata, the pyramidal fibers above the decussation may act upon the cells of origin of other efferent systems. The pyramidal fibers may act indirectly upon motor neurones in the red nucleus through intercalated neurones arising in the thalmic nuclei and, probably also, directly upon cells in the red nuclei through collaterals. There are also pyramidal fibers terminating about motor cells in the reticular formation, and one should not lose sight of the possibility that the pyramidal fibers may also have some indirect connection with the cells of the substantia nigra. (Figure 1.) Which of these possibilities will be shown to be anatomical and physiological facts can be told only by future work. To quote from Rademaker and Winkler:[11] "Such researches may have a great interest for: 1, a physiology of the higher parts of the central system, wanting to be freed from the psychological nomenclature in which it is, necessarily at present, bound; 2, an anatomy wanting to know the systematic architecture of those higher parts, which till now, is only known very roughly."

When these possibilities of the connections of the pyramidal fibers with the motor neurones of the so-called "extra-pyramidal" system are considered, perhaps one should not be wholly dismayed or greatly surprised by the fact that the effects of bilateral motor

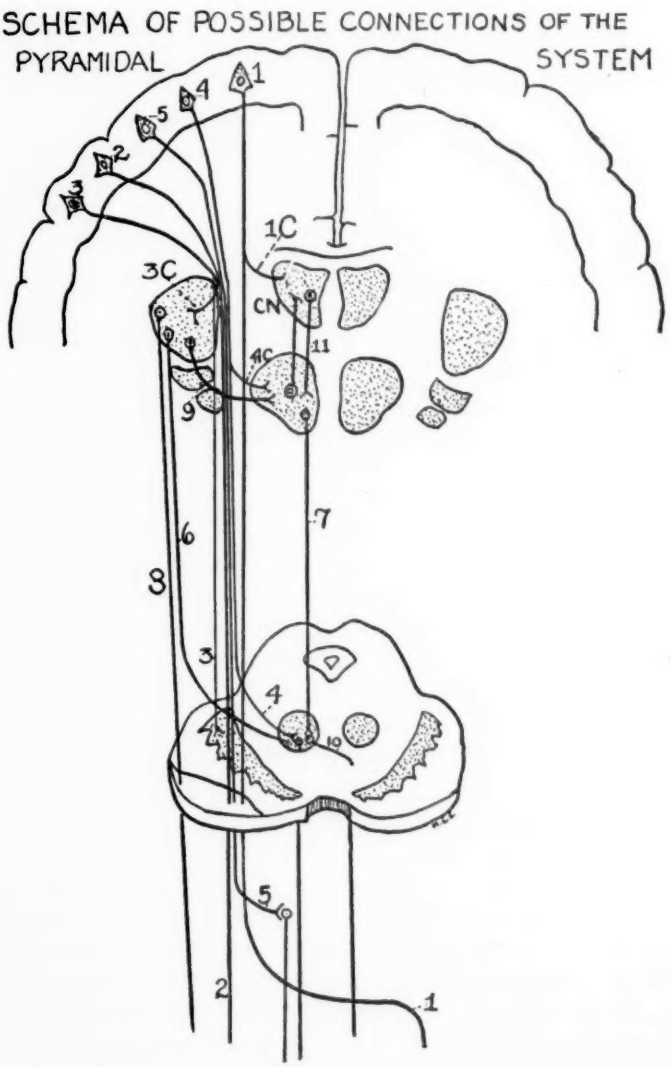

FIG. 1.—Schema of possible connections of the pyramidal system. (It must be remembered that not all of the possibilities shown in the scheme have been demonstrated anatomically.) 1, 2, 3, 4, 5, cortical cells of origin of pyramidal fibers. 1, Cell or origin of a pyramidal fiber sending a collateral 1C to the caudate nucleus, CN, and crossing in the decussation at the lower end of the medulla. 2, Cell of origin of direct pyramidal fiber. 3, Cell of origin of pyramidal fiber giving off collateral 3C, to the globus pallidus and terminating about a cell in the substantia nigra. 4, Cell of origin of pyramidal fiber 4, terminating in the red nucleus. 5, Cell of origin of pyramidal fiber 5, terminating about a cell in the recticular formation. 6, Fiber originating in globus pallidus and terminating in the red nucleus. 7, Fiber originating in the thalamus and terminating in the red nucleus. 8, Uncrossed fiber originating in the globus pallidus (Morrison). 9, Fiber originating in the globus pallidus and terminating in the thalamus. 10, Crossed fiber of rubro-spinal tract. 11, Fiber originating in the caudate nucleus and terminating in the thalamus.

area ablation, following after an interval of recovery from median incision of the pyramidal decussation, are so severe. There is also the possibility, suggested by Balighian's [13] experiments, that the actual crossing of pyramidal fibers in the decussation occurs over a wider extent than is indicated by the anatomical findings. It should be possible to check this histologically. We will return to this point a little later, when considering combined lesions of the pyramidal and rubrospinal systems. But the possibilities which we have pointed out should be sufficient to arouse suspicion as to the validity of the assumptions of the rigidity of separation and high degree of independence of function of the pyramidal and so-called extra-pyramidal, motor mechanisms. The type of convulsions elicitable immediately after ablation of the cerebral motor cortex following median, longitudinal incisions of the pyramidal decussation is tonic. If one motor cortex is removed, tonic convulsions appear on one side of the body and clonic upon the other. Judging by the motor effects and the type of convulsion elicitable when one or both motor cortices are removed at some interval after median longitudinal splitting of the pyramidal decussation, the corpus striatum, thalamus, red nucleus and other lower motor mechanisms have not been the sole mechanisms of compensation for injury to the pyramidal decussation.

2. LESIONS OF THE MIDBRAIN AND PARTICULARLY OF THE RUBRO-SPINAL DECUSSATION.

Most of the discussion of the function of the lower motor mechanisms, striatal, thalamic and mesencephalic, even reticular, has been based upon the deportment of animals after partial or complete elimination of the cerebral hemispheres, or any and all structures above the ones whose function is to be determined. The argument has been advanced that, since compensation may occur for injuries to the pyramidal system, the lower motor mechanisms bear the great burden of motor function in intact animals. It seemed of importance, therefore, to devise some method of eliminating some one or more of these lower motor mechanisms without injury to the pyramidal system. Such a procedure might give us an entirely different outlook upon the function of these lower motor mechanisms. We have, accordingly, made a median longitudinal incision of the midbrain in such a way as to sever, along with other

structures, the decussation of the rubro-spinal tracts (decussation of Forel).

A. The deportment of animals after median longitudinal incision of the decussation of Forel. The immediate effects of this procedure are sometimes a severe prostration and grave motor disability. The animal is unable to rise or stand for some days, and, if the incision passes a little to one side of the median plane, it may lie upon one side to the exclusion of the other. We have, however, seen an animal rise and walk within two or three hours. One pupil may be much more dilated than the other, due to the greater involvement of the third nerve nucleus of one side. There is sometimes, also, a torsion of the head with the chin turned to the side of the wider pupil. At the end of two weeks, the animal may be walking around very well. There is sometimes some spasticity of all the limbs, which may be raised higher than normal in walking, after the manner of the goose-step. Such a condition may persist for months. Judged by the same criterion that has been applied for the estimation of the function of the pyramidal system, one would be perfectly justified in concluding that the rubro-spinal system had a very slight function under normal conditions, since motility returns. Either our estimate of the function of the pyramidal system is incorrect, or else our estimate of the rubro-spinal system is incorrect. Possibly both estimates are incorrect.

The type of convulsions elicitable within a few hours after incision of the rubro-spinal decussation, or after an interval of weeks or months, is clonic. The tonic convulsions are either wholly suppressed, particularly in the early period after operation, or appear in diminished degree, and the lethal dose of absinthe is sometimes greatly increased.

Median longitudinal incision of the midbrain injures other structures than the decussation of Forel. The posterior commissure and Meynert's decussation are injured, and any commissural fibers between the superior colliculi would be severed. If the lesion passes a little to one side of the median line, the nucleus of one third cranial nerve is injured. We have not found deafness as a result of this lesion. The picture is, therefore, that of a lesion of the decussation of Forel plus some other structures, and the actual effect due to the lesion of the decussation of Forel alone would probably be somewhat less than the total effects observed.

Animals with a lesion of the midbrain have been more susceptible to the affections of the respiratory tract—sneezing, infections of the nasal sinuses, etc.—than control animals or animals with other cerebral lesions, during the winter, and a number have died some weeks or months after the operation. One has apparently recovered from an infection of the frontal sinuses after a convalescence of about six weeks. This high mortality from extraneous causes has reduced the number of observations made below what we had hoped for.

The results so far indicate that tonic convulsions fail completely, or are greatly reduced, in the early post operative period, while clonic convulsions are immediately obtainable in undiminished intensity. If the rubro-spinal system had any close relation to the genesis of clonic convulsions, we would expect some decrease in their intensity at first. There is an indication, also, that the tonic element in convulsions increases in intensity during the period of recovery, but we have no accurate information at present on the source of this tonic element, nor of the full intensity which it may finally attain.

3. COMBINED LESIONS OF THE MIDBRAIN AND OTHER SYSTEMS.

The great number of possibilities of combined lesions of two or more structures in the nervous system makes dependence upon memory alone unreliable, and some form of record becomes necessary. We have adopted the form of record on page 582 for each individual structure of which lesions have been made. It shows just what combinations of lesions have been made, and has space for a record of a third lesion in case any such is made.

The question as to the effect of a second lesion of some other system upon an animal which has recovered from median longitudinal incision of the midbrain suggests, first of all, a study of combined lesions of the rubro-spinal and pyramidal systems.

A. Median longitudinal incision of the midbrain followed by a median longitudinal incision of the pyramidal decussation. The fact that the recovery of an animal is more complete after splitting the pyramidal decussation than after bilateral motor area ablation led us to study this case first. Such a combined procedure should afford the basis for a comparison of the motor power of the uncrossed pyramidal system when all other systems are intact, with

Structure of which first lesion is made *decussation of Forel*

Number on chart *80* Lesion unilateral ..... side; bilateral *median incision of midbrain*

1.	17.	33.	49.	65.	81.	97.
2.	18.	34.	50.	66.	82.	98.
3.	19.	35.	51.	67.	83.	99.
4.	20.	36.	52.	68.	84.	100.
5.	21.	37.	53.	69.	85.	101.
6.	22.	38.	54.	70.	86.	102.
7.	23.	39.	55.	71.	87.	103.
8.	24.	40.	56.	72.	88.	104
9.	25.	41.	57.	73.	89.	105.
10.	26.	42.	58.	74.	90.	106.
11.	27.	43.	59.	75.	91.	107.
12.	28.	44.	60.	76.	92.	108.
13.	29.	45.	61.	77.	93.	+ = increase of effect.
14.	30.	46.	62.	78.	94.	− = decrease of effect.
15.	31.	47.	63.	79.	95.	0 = no change in effect.
16.	32.	48.	64.	80.	96.	when second lesion is made.

Followed after an interval of recovery by a second lesion of

Number	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
Effect												
	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.
	25.	26.	27.	28.	29.	30.	31.	32.	33.	34.	35.	36.
	37.	38.	39.	40.	41.	42.	43.	44.	45.	46. ±	47.	48.
	49.	50.	51.	52.	53.	54.	55.	56.	57.	58.	59.	60.
	61.	62.	63.	64.	65. +	66.	67.	68. +	69.	70.	71.	72.
	73.	74.	75.	76.	77.	78.	79.	80.	81.	82.	83.	84.
	85.	86.	87.	88.	89.	90.	91.	92.	93.	94.	95.	96.
	97.	98.	99.	100.	101.	102	103.	104.	105.	106.	107.	108.

Second lesion number ..... *68* *65 (unilateral)*

Followed by third no. ..... *65* *46 (unilateral)*

Effect of third lesion ..... + +

Remarks: 80 followed by 46; torsion of head reversed when Scarpa's ganglion of side opposite to that of deviation of vertex of head is removed; increased when Scarpa's ganglion of same side is removed.

the conditions seen when some other system has been injured. There is no reason for supposing that median incision of the midbrain has interfered with the direct pyramidal fibers to any greater extent than with the crossed. If, therefore, the recovery of an animal after splitting the pyramidal decussation, all other systems being intact, is due entirely to the uncrossed pyramidal fibers, the results of splitting the pyramidal decussation should be the same in animals in which the midbrain is intact as in animals in which the midbrain has been split longitudinally. If, however, the recovery of an animal after an incision of the pyramidal decussation, all other motor systems being intact, comes about through some effect of the pyramidal fibers above the decussation upon the cells of origin of other systems of efferent fibers, the results of splitting the pyramidal decussation after an injury to some other so-called extra-pyramidal motor system should be measurably more severe than when all other motor systems are intact.

The immediate results of a combined lesion of both systems on the same day are severe. The animal lies on its side unable to rise, but there may be rhythmically alternating movements of the limbs, particularly of the forelimbs. Movements of the head are still possible. On injection of absinthe, some clonic response, particularly in the forelimbs, head, face, eyes, ears, is obtained. Tonic convulsions are long delayed or absent.

When the pyramidal decussation is split after an interval, the animal shows at first a severe motor disability, but again begins to move about after an interval of a few days. The degree of recovery is noticeably less than after splitting the pyramidal decussation when all other motor systems are intact. After an interval of a month, the feet slip about on a smooth floor and let the animal down. It is our general impression that the motor disability of an animal at this stage of recovery, is really noticeably greater in some respects, than in an animal with bilateral ablation of the cortical motor areas at a similar stage of recovery. It is much greater than the motor disability seen after simple median longitudinal incision of the pyramidal decussation.

B. Median longitudinal incision of the midbrain with ablation of the cortical motor areas. When both lesions are made at the same time, the effect is extremely severe. The animal lies on its side without even lifting its head from the floor, and usually makes no

sound. Some movements of the limbs are occasionally seen, but the are not of any great extent. Convulsions are uncertain and difficult to elicit under absinthe.

When ablation of the cortical motor areas is made after an interval, the effects are again of extreme severity and, so far, we have no animal which survived this second operation more than a few days. It lies in almost any position on the floor, sometimes getting its feet outstretched beneath it, but unable to rise or walk. The forelimbs are sometimes outstretched to either side, with the whole of the mesial surface applied to the floor. The animal does not appear to have lost the subjective side of orientation, if one may speak of it in such a way, and even has some power of getting itself into an upright position, after being turned on either side or on its back. Evidently, following the lesion of the rubro-spinal tract, the animal has been able to compensate exceedingly well for the loss of the red nucleus and its crossed fibers in matters of orientation and righting itself. We may express the opinion that other mechanisms aside from the rubro-spinal are normally concerned in the process of orientation.

Two animals have survived unilateral motor area ablation after median incision of the midbrain, while one has succumbed within a few days.

C. Median longitudinal incision of the midbrain with median longitudinal incision of the pyramidal decussation and cortical motor area ablation at successive intervals. The extreme severity of the effects of motor area ablation following midbrain lesions led us to try a more gradual approach to the elimination of the pyramidal and rubro-spinal systems. In an animal which has recovered successively from median incision of the midbrain and splitting the pyramidal decussation, one cortical motor area was removed. The prostration was extreme and the animal succumbed in a few days without regaining the power of locomotion. The limbs on the side corresponding to the motor area ablation would give way and let the animal down when it tried to rise. The effects seemed to be out of all proportion to those seen after simple unilateral motor area ablation, either with or without previous splitting of the pyramidal decussation where the rubro-spinal system is intact. It would appear from these results that the pyramidal system has other efferent outlets than the rubro-spinal system after splitting the decussation

of the pyramidal tract. For the number of uncrossed pyramidal fibers eliminated by cortical motor area ablation following splitting of the pyramidal decussation is independent of any median lesion of the midbrain. The difference in the effect of cortical motor area ablation, after an interval of recovery from splitting the pyramidal decussation, when the midbrain is intact as compared with the effect of cortical motor area ablation when the midbrain is split longitudinally cannot be due solely to a difference in the number of uncrossed pyramidal fibers which are eliminated under the two different conditions. Nor can the greater severity of the effects of median incision of the pyramidal decussation after previous median incision of the midbrain, as compared with the effects observed when all structures above the pyramidal decussation are intact, be due solely to a difference in the number of pyramidal fibers injured under the two different conditions. The most logical inference from the facts is that the remaining fibers of the pyramidal tract have more indirect efferent outlets when the rubro-spinal system is intact than when it is injured; and that, after injury to the cortical motor cells, the lower motor mechanisms may compensate for cortical loss more completely and more speedily when the rubro-spinal system is intact than when it is injured. The converse of this proposition would also appear to hold, namely, that compensation for the loss of the rubro-spinal system is more speedy and more complete when the cortical structures are intact than when they are absent. The cortical motor area would appear, also, to have other indirect efferent outlets than through the rubro-spinal system, after splitting the pyramidal decussation.

The deportment of animals after ablation of the cortical motor areas and median longitudinal incision of the midbrain calls attention to the remaining motor mechanisms. The procedure of combined lesions eliminates two of the mechanisms which, on anatomical grounds, we have come to regard as important motor mechanisms. We have even insisted, possibly on insufficient grounds, that they belonged to two different motor systems, as is implied in our terminology of pyramidal and extra-pyramidal mechanisms. When we look at what is left of the motor systems, we find the corpus striatum, the motor portions of the thalamus, the substantia nigra and the motor nuclei in the reticular formation, together with the phylogenetically old short pathways in the me-

dulla and spinal cord, remaining anatomically intact so far as any direct injury is concerned. Phylogenetically, the red nucleus is first found in fishes (nucleus reticularis superior of Raja clavata) as the most anterior of the reticular motor nuclei.[14] The character of the red nucleus appears to change in vertebrate development, and there are other nuclei in mammals which appear to have more of the characteristics of the primitive reticular nuclei. The paleostriatum, the old portions of the thalamus, the reticular nuclei and the tracts descending from them, together with the short descending tracts in the spinal cord, constitute the motor system of the fish. Taken altogether, it is a very efficient system. On the basis of anatomical relationships alone, even though the character of the red nucleus changes in the evolutionary development from fish to mammal, it might be argued that median longitudinal incision of the midbrain should render a fish as helpless as a cat after ablation of the cortical motor areas and incision of the midbrain decussations. But the phylogenetically old motor mechanisms in fish seem more adequate for their task than they are in mammals.

4. LESION OF THE RUBRO-SPINAL DECUSSATION AND UNILATERAL EXTIRPATION OF THE VESTIBULAR PORTION OF THE EAR.

It has been mentioned that, in some animals, in which the longitudinal incision of the midbrain was a little to one side of the median line, there was a marked difference in the diameter of the pupils of the two sides, with some torsion of the head, the chin being directed to the side of the wider pupil. Unilateral extirpation of the vestibular portion of the ear was done in two such animals, in each of whom the left pupil was the wider, and the vertex of the head turned to the right.

1. Extirpation of the left vestibule. The torsion of the head changed with the vertex to the left, as is usual after vestibular lesions. The animal soon recovered its motor facility and walked about the room in its usual manner.

2. In a second animal (experiment number 569), in which the vertex of the head was turned to the right, following median incision of the midbrain (Oct. 24, 1929), the left cortical motor area was removed after an interval (Nov. 12, 1929). The animal regained the ability to walk about and, after a second interval, the right vestibule was extirpated (Jan. 16, 1930). The torsion of the

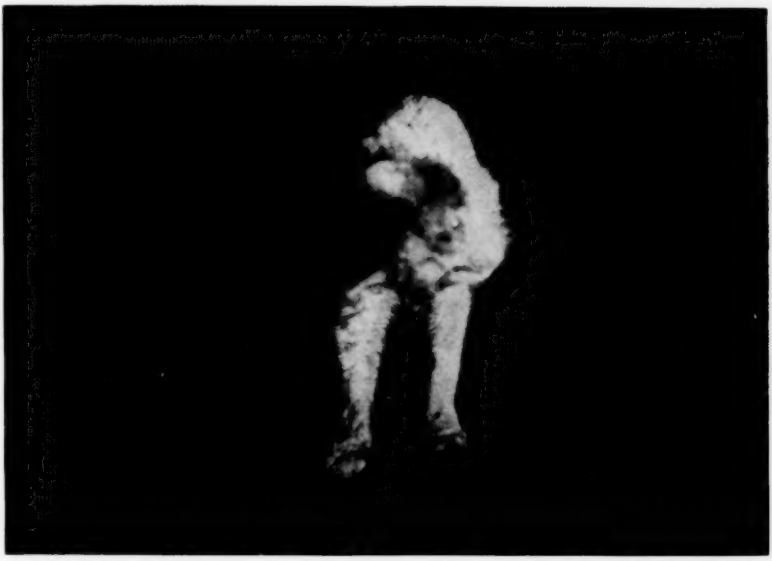

FIG. 2.—Cat of experiment number 569. Median longitudinal incision of the midbrain, followed, at intervals, by ablation of the left cortical motor area and extirpation of the right internal ear. The vertex of the head is turned strongly to the right and the nose is on a level with the occiput, with the left ear pointing directly upward.

head to the right was so far increased that the chin was at times actually turned up into the air, being higher than the vertex of the head. The appearance of this animal standing head-on to the camera, is shown in Figure 2. Absinthe was injected intravenously Jan. 29, 1930. Tonic convulsions appeared on the right side of the body, with clonic convulsions appearing at larger doses. The tonic element was never very well marked. There were the usual clonic convulsions on the left side.

Median longitudinal incision of the midbrain and bilateral ablation of the cortical motor areas gives a condition which, so far as any motor mechanisms are concerned, is more than equivalent to decerebration. Evidence so far accumulated by a number of investigators tends to show that the rubro-spinal system is a more adequate motor mechanism in cats than the striate and thalamus taken together. The decerebrate animal has neither corpus striatum nor thalamus, while the animal with the combined motor area and midbrain lesions has both corpus striatum and thalamus, although the efferent pathways from these two structures may, in part, be blocked by the lesion of the midbrain. There are, however, as Morrison [15] has shown, uncrossed descending fibers from the corpus striatum. The whole central sensorium is, however, preserved almost intact, and there is no decerebrate rigidity. The animal lies with feet outstreetched in a flaccid condition, although the supposed inhibitory effect of the pyramidal system is completely lacking, rising on its feet at rare intervals and then falling helpless to one side or the other. The conditions in the animals with combined motor area and midbrain lesions must, therefore, be considered in all discussions of the genesis of decerebrate rigidity.

Further work along this line affords a promise of a more firmly grounded and more adequate explanation of so-called decerebrate rigidity than we now have. The persistence of spasticity in some animals after median incision of the midbrain, the pyramidal system being intact, in itself casts grave doubt upon the hypothesis that decerebrate rigidity develops because of the removal of the supposed inhibitory influence of the cortical motor cells upon the lower mechanisms. Other grave objections to the hypothesis of inhibition may also be adduced.

The facility of animals for maintaining the upright position at all times after median longitudinal incision of the midbrain may

seem surprising to those who hold that orientation and the righting reaction are related only to the rubro-spinal system. But one should remember that, while a lesion of the red nucleus may completely abolish the righting reactions when all higher motor mechanisms are absent, a lesion of the rubro-spinal system may not necessarily abolish the righting reactions where all the other motor mechanisms are intact. The conclusion that the rubro-spinal system is the sole central mechanism for orientation and the righting reaction rests upon an incomplete and insufficient analysis of the central mechanism. What the other parts of the righting mechanism may be is still unknown.

The work so far has dealt mainly with two systems—the pyramidal and rubro-spinal—leaving the striate and thalamus virtually untouched. We do not know, for instance, to what extent the striate and thalamus may be concerned in the recovery of clonic convulsions after pyramidal injuries, or whether the rubro-spinal and reticular systems are sufficient in themselves for the genesis of clonic convulsions if a sufficient interval of recovery is allowed. Nor are we clear as yet on the degree to which tonic convulsions may return if a long interval of recovery be allowed after median incision of the mid-brain. The striate, thalamus, substantia nigra and recticular nuclei seem to have some rôle in the production of tonic convulsions, but we do not know which of these structures are concerned, nor the degree in which each is competent. A more exhaustive experimental analysis is needed. It seems clear, however, that, after injury to any one part, or even more than one part, of the motor system, the remaining parts will carry on.

We may now consider the bearing of these experimental results upon some of the current views on the functional organization of the motor mechanisms of the central nervous system. According to the point of view which one holds, the regression of symptoms and the return of motor facility after a lesion of any part of the motor mechanisms may be due to any one of these possible processes, namely:

1. The subsidence of inhibitory and shock effects which result from the injury (Goltz).

2. The increased functional activity of some associated system or pathway after injury to the first (Hughlings Jackson).

3. The vicarious assumption of the function of the injured portion by some other mechanism which previous to the lesion had no functional relation to the injured portion.

(1) If recovery is due to the regression of shock or inhibition, no two successive lesions should have any greater final effect than any other two, since the shock effect of any one extensive injury of the nervous system should be as great as that of any other. Since the actual trauma is the same in each case, the effects of splitting the decussation of the pyramidal tract should be the same:

(a) When the motor cortex is intact.

(b) When the motor cortex has been ablated.

(c) When the decussation of the rubro-spinal system has been severed. Such a conclusion is at variance with the facts.

(2) If the recovery from a lesion of one part of the motor system is due to an increased functional activity of the remaining part, a particular pair of successive lesions might be expected to have a much greater total effect than some other pair of successive lesions. For example, splitting the pyramidal decussation followed by ablation of the cortical motor areas has a greater effect than splitting the pyramidal decussation and removal of a considerable portion of the parietal region of the cerebral cortex. The motor effects of ablation of one cortical motor area are not noticeably increased by removing subsequently practically all of that portion of the cerebral hemisphere of the same side lying above the lateral ventricle.[16] It is not mere mass of tissue which is removed, nor indiscriminate pairing of successive lesions which is significant, but the particular grouping of lesions. The close relationship between the effects of a second lesion of some structure which, on independent grounds we have reason to believe has motor functions, and the intensity of the supposed shock effect, on the one hand, and the absence of any such relationship where the second lesion is of some structure which we have no independent reason to believe has any motor function, shows, that, if shock is a real thing at all, it is a capricious thing following no known and, apparently, no discoverable laws. On the basis of Jackson's view of the change in the quantity of nervous energy passing over the remaining pathways after injury to one or more, the close relationship of the severity of effect of the second lesion to its known motor functions is understandable, and is to be expected.

(3) The possibility of vicarious assumption of function has been sufficiently excluded by the discussion under (1) and particularly (2) above. The mere fact that a particular pair of lesions has a much greater effect than some other pair, even though there may be one structure common to the two pairs, shows that indiscriminate compensation for a given lesion does not occur.

We may reexamine in the light of more recent results Munk's " conception of " Isolations-Aenderungen " or the changes that occur in an isolated portion of the nervous system. Munk's conception was more or less limited to conditions in the isolated portion of the spinal cord after total transection, and it may not be strictly applicable to the conditions of our experiments, in which there is no isolated portion of the nervous system. Sherrington has used isolation dystrophy to express this idea of a retrogressive change. The general discussion of the effects of lesions of the cortical motor areas to be found in the literature embody, however, much the same idea which Munk apparently had in mind, namely, that no changes of a progressive type occur in the remaining systems.

There is, of course, degeneration of that portion of the neurone which is separated from its cell body in spinal and cerebral lesions alike. Sometimes, also, the cell of origin may be affected, though seldom to the point of degeneration. In young animals, the next neurone in the chain on the afferent side may fail to develop. There may be a chromatolysis of the next neurone in the efferent chain, but no actual degeneration. Fibrillary twitching and degeneration of the skeletal muscles is not seen in the conditions under consideration. Some " Isolationsaenderungen " of a retrogressive type—some isolation dystrophy, does occur. Goltz and his school would hold that the return of reflexes or of movement after a lesion is due tc the gradual passing away of the shock effect due to the lesion itself. On Jackson's view there would be a change in the quantity of nervous energy passing over the remaining pathways. In addition to the retrogressive dystrophic changes, there would be other changes of a compensatory or progressive nature.

Compensatory changes of a progressive nature are so well known in various organs of the animal body as to excite no comment at present. Every physiologist recognizes that one kidney does more work after the other one is removed. The failure of the patient to show grave symptoms after loss of one kidney is no longer held

to mean that the lost kidney had no function. We are familiar, also, with the hypertrophy of the heart after the development of valvular insufficiency or mitral stenosis. We no longer say that, because there are no grave symptoms after removal of the thymus or the spleen, these glands have no function. Only in the nervous system are structures supposed to have no function if the animal recovers so far that no grave symptoms are apparent. And only in the nervous system is it said so emphatically that no changes of a progressive nature occur by way of compensation for injury. All of which is not only at variance with the known changes in other systems, but lacks any basis of fact in the nervous system itself.

5. OBSTRUCTION TO THE VENOUS OUTFLOW FROM THE BRAIN.

The median incision of the decussation of the rubro-spinal tracts does not, so far as we are aware, interfere with any vascular reaction, either within the central nervous system or elsewhere. Attention has been directed within recent years to the vascular element in the genesis of epileptic seizures. Henderson and Gillespie [18] have cited some earlier work by Professor G. N. Stewart and one of us (F. H. P.) in support of the view of a vascular genesis of epileptic convulsions. The obstruction of the venous outflow of the brain supplements the observations on temporary interruption of the cerebral arterial supply, and it affords, also, a basis of control for some projected lesions of the structures of the posterior fossa.

A. Obstruction to the venous outflow from the brain was accomplished in two ways. In one animal the external and internal jugular veins of both sides and the vertebral vein of one side were ligated at one time. After a short period, in which there was some bulging of the eye on the side of the ligated vertebral, and some slight sluggishness of deportment, the animal recovered without noticeable symptoms. It was kept for a year or more during which time it increased in size and weight. At the end of this time, absinthe was injected intravenously. The minimal convulsive dose was low, as was also the lethal dose. The convulsions were clonic in type. No spontaneous convulsions were ever observed in this animal.

B. Obstruction to the venous outflow from the brain was accomplished in a series of animals by blocking both transverse

sinuses. The muscles were freed from the cephalic and caudal aspects of the lambdoidal ridge of the occipital bone for about two centimeters on each side of the median line, and the temporal muscle was freed from its attachment to the parietal ridge to about 5 millimeters rostrad of the attachment of the tentorium to the inner surface of the parietal bone. A trephine opening was made on each side in front of the lambdoidal ridge, but cutting across the line of attachment of the tentorium. On removing the disc of bone, the tentorial vessels were stopped up with bone wax, the trephine opening was enlarged with rongeurs through the lambdoidal ridge of each side, and the rather free hemorrhage from the transverse sinuses stopped with bone wax. Sometimes the portion of the parietal and interparietal bones lying between the trephine openings was also removed with the rongeurs, and all bleeding stopped with bone wax. The median longitudinal sinus is, of course, blocked by any procedure which obstructs all the lateral sinuses. Only slight or inconsequential injury need be done to the superior surface of the vermis cerebelli. It is not necessary to injure the dura. The muscles are then sutured together and the skin incision closed.

The immediate effects of the operation are more serious than can be explained on the basis of direct injury to the cerebellum. For some hours, the animal may not rise or stand, and may make very few movements of any kind. Intravenous injection of absinthe within a few hours elicits clonic convulsions at a significantly lower dose than in normal animals. In one cat (experiment number 591), in which a considerable quantity of bone wax was pressed down through the opening in the bone, so that it compressed the left side of the vermis and the median portion of the left lateral lobe of the cerebellum, the clonic convulsions appeared at a low dosage, and no marked tonic convulsions occurred even at the lethal dose, which was low. The convulsions observed immediately before death were clonic. Ophthalmoscopic examination before the injection of absinthe showed retinal hæmorrhage in the left eye.

Some of the animals were allowed to live for varying periods of time, but they remained sluggish and less alert than other animals with more extensive destruction of brain tissue; but none of them showed what we considered as typical signs of direct cerebellar involvement. The motor symptoms were not those of cerebellar

lesions. In one animal, in which the transverse sinuses alone were blocked, without invasion of the tentorial vessels, no difference in deportment was observable after the first four or five days. The animal now seems alert and active as any other cat. Apparently, although the vessels in the tentorium are small, blocking them in addition to the transverse sinuses adds to the gravity of the symptoms. In all the animals so far tried, after an interval of recovery, following blocking of the sinuses, intravenous injection of absinthe has elicited clonic convulsions at a significantly lower dosage than normal and the lethal dose has also been low, sometimes even below the minimal convulsive dose for intact animals.

The compensation for obstruction of the lateral and median longitudinal sinuses in the cat is greater than in the human, where such obstructions to venous outflow are speedily fatal.

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THE CENTRAL MECHANISM OF GENERALIZED EPILEPTIC FITS.*

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Epilepsy is a problem as old as medicine or even older. One has tried to study and to solve it from many different points of view, by simple clinical observation, by anatomical researches, by animal experiments, and in the last years especially by the study of metabolism. We have learned the importance of chemical factors, of oxygen supply, of ionic equilibrium, of acid-base relationship, of the products of endocrin glands, but no disorder of metabolism has been found which would be specific for epilepsy. Lennox and Cobb,[1] in their last excellent review, come themselves to the conclusion that anoxæmia, alcalosis and similar pathological changes are only contributory factors, tending to increase the irritability of nerve tissue, but producing seizures only in those who are subject to seizures.

During this period of study of metabolism, the analysis of the central mechanism of the fit itself has been rather neglected, but we have to remember that we have also in this field many open questions, the genesis and localization of the clonic and of the tonic part of the fits, the mutual relation of these two components and of the parts of the central nervous system where they are localized, the ways of the impulses within the central nervous system between the different areas of the cortex and between cortex and sub-cortex. Animal experiment seems to bring a considerable help for the study of these questions; it is possible to produce in the usual laboratory animals not only in apes, but also in dogs and cats by electric stimulation, by local application of toxins on the cortex, like strychnin, or by general poisoning like by cocaine, absinth, thujon fits which are very similar to human epilepsy. It is very striking how similar the central motor mechanism of man, apes and quadru-

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peds react to normal stimuli. This similarity shows that the organization of this apparatus follows the same plane in different groups of animals. On the other side let us not forget (compare also Trendelenburg [9]) that the picture of rhythmic motor phenomena as we find it in lower animals is often only similar to human epilepsy, that much criticism is necessary before we draw conclusions from such observations in animals to the mechanism of human epilepsy. An example may explain it. Brown-Séquard observed in guinea pigs some time after cutting the sciatic nerve tonic and clonic fits following mechanical stimulation of the skin. These fits have been studied afterwards by many authors who draw conclusions to the pathology of human epilepsy until Graham Brown [3] showed that these fits have nothing to do with epilepsy and are only a form of the scratch reflex.

We have to distinguish, as you know, in the clinical picture of epileptic fits a clonic and tonic component. Much work has been done to analyze from which part of the central nervous system these two components take their origin. We may say that the majority of these experiments leads to the conclusion that the *clonic component* is of cortical origin as it was first stated by Ziehen [2] (for instance Rothmann,[4] Morita,[5] Trendelenburg [9]). Some experiments seem contradictory. Schoen,[7] for instance, found that the convulsions following hexeton and cardiazol poisoning had the same type in normal rabbits and in animals without forebrain. These movements are not true clonisms, but running movements, as Trendelenburg [6] and Bertha [8] have shown.

Much more striking than the experiments of Schoen are these of Pike and Elsberg.[6] They found some weeks after extirpation of the motor area of the cortex clonisms following absinth poisoning. But we have to remember that the lower centers get, some time after the destruction of their connections with the higher centers, special qualities under the influence of their isolation as Munk has shown for spinal cord reflexes. The spinal cord itself has the possibility to answer a constant stimulus by rhythmical discharges. The scratch reflex of the spinal dog is the best example for this. It seems that such isolated centers get also the possibility to respond to toxic stimuli by rhythmical phenomena. But it does not prove that clonic phenomena take their origin in sub-cortical centers under normal conditions. If the higher centers send the normal

impulses to the cord, the clonic component of the epileptic fits seems to be of cortical origin.

Here a new question arises. If we innervate the skeletal muscles from the cortex by voluntary impulses, we get a series of impulses following so quickly that the mechanical effect looks continuous (tonic). We find the same if the motor area of a man or a mammallian is stimulated by slight faradic stimuli; why do we get quite a different form of discharges, a rhythmical interruption of the impulses sent out by the cortex in pathological conditions or following stronger or repeated cortical stimulation? The simplest explanation for rhythmical phenomena is as you know the refractory period, the short period of loss of excitability following each impulse, which is shown not only for the heart but also for skeletal muscles, nerve fibres, reflexes. For the cortex we know since the experiments of Broca and Richet [10] that there exists also a refractory period. The phenomenon of the refractory period alone is not sufficient to explain the clonic convulsions following cortical stimulation under special conditions (see also Muskens [11]). It is a normal phenomenon which does not explain the difference between normal and pathological forms of reaction. To understand these differences we have to remember the ideas which have been developed during the last years on the mechanism of excitation (compare Spiegel [12]). Following Nernst's theory excitation is the consequence of a change of ion concentration on semi-permeable membranes. The normal cell has semi-permeable walls, the kations may go through these walls, the anions cannot pass through. Every form of stimulation, for instance the electric current, brings a movement in these ions; as only some of them can pass through the membrane, others are not able to do it, changes of concentration are the consequence. These changes are not without influence on the membrane itself, they usually destroy or injure the membrane at this place, lower here its semi-permeable qualities, as it has been shown on muscles and nerves. One may say that the membrane gets a hole so that all ions may pass through. The action current is the most striking sign of this injury. The refractory period is another consequence of it. It lasts as long as the membrane needs for its recovery. If we stimulate the cell for a longer time with strong stimuli finally the membrane cannot recover as quickly as before, a longer pause develops, until it gets again its semi-permeability and a new ion concentration may develop.

Such phenomena can be observed even in peripheral nerves. Brücke and Field found here that the refractory period becomes longer after repeated stimuli. And indeed, if one stimulates a peripheral nerve with a stronger constant current for some time, one may observe rhythmical phenomena, as Garten, Ebbecke have shown by the study of the action currents. I mention these facts as they probably show us a way for the further study of clonic convulsions. We may suppose that similar changes like these observed on peripheral nerves may occur in ganglion cells, following repeated stimulation. Under pathologic conditions there may be only a difference by degree. Even a slight stimulus may now be able to cause a strong discharge of the cell. The semi-permeability of its membrane may be easier destroyed or need longer time for its recovery so that the discharges of the cell do no more follow each other quickly but at intervals and the mechanic effect on the muscle is the clonus.

The theory of which I can give here only a short outline is supported by some facts. It seems that such drugs or influences which increase the readiness of the cortex to react with epileptic fits, increase the permeability of membranes (compare Georgi [12]); influences which are able to make the membranes more dense, lower also the epileptiform reactivity of the cortex (for instance, X-rays).

It may be that also in the interesting experiments of Syz,[14] the change of the permeability of nerve cells played a rôle. You will remember that he found in frogs following brain injury or asphyxia that a convulsant dye entered the brain substance more readily. He draws the conclusion from these experiments that especially the permeability of the barrière hémato-encéphalique was increased. It may be that in these experiments for instance by asphyxia not only the permeability of this barrière but also of the nerve cells themselves was increased. In any case, the study of the permeability of the nerve cells which I intend to report in detail in a next paper seems a way not only for a better understanding of the rhythmical phenomena of cortical discharge but also to find new methods for therapy.

The second component of epileptic fits is, as you know, the *tonic part*. To understand it, it may be advisable to recall the normal mechanism of tonus innervation as all pathological phenomena are nothing else than changes of normally pre-existing mechanisms.

Tonus is a reflex phenomenon; the following reflex arcs maintain the tonus of skeletal muscles (compare Spiegel[15]): first, a short reflex arc having its center in the spinal cord and its origin in the proprioceptive nerves in the skeletal muscles themselves; second, a long reflex arc having the same origin but its center in the subst. reticularis of the rhombencephalon; third, a reflex arc starting from the labyrinth and whose centers are the vestibular nuclei in the medulla oblongata and in the pons. We know by Sherrington's decerebration and by the further analysis of decerebrate rigidity (Magnus,[15a] Rademaker,[15b] Spiegel[15] and others), that the central nervous system cranial from the rhombencephalon is not necessary to maintain the tonus of skeletal muscles. The same is true for the cerebellum. These parts of the nervous system regulate only by inhibitory or stimulating impulses the tonus of skeletal muscles which is maintained by the three reflex arcs mentioned above.

The importance of the rhombencephalic centers for the genesis of tonic spasms is shown by different experiments. As there exists some relations between epilepsy and tetany, it may be advisable to mention experiments in which we tried, in collaboration with Nishikawa,[16] to analyze the mechanism of the spasms which develop after extirpation of the parathyroid. It was shown that these spasms like decerebrate rigidity may be produced even if the Nucl. ruber or the Tractus rubrospinalis is destroyed on both sides. Such spasms were observed also after a nearly total extirpation of the cerebellum but they are not observed if the spinal cord is no more in connection with the higher centers (Lanz,[16a] Biedl,[17] etc.). We may draw the conclusion that the tonic spasms following the extirpation of the parathyroids are the product of reflexes which have their center outside the cerebellum between the Nucl. ruber and the spinal cord.

In epilepsy the rhombencephalic centers may be directly stimulated by toxins as it is shown by experiments in which the tonic component of epileptic fits was observed in decerebrated animals, for instance, after injection of absinth, cocaine (Fuchs[18] and others), but there is not only a direct stimulation of the rhombencephalic centers possible. They may be reached and excited even by cortical impulses, for instance, in traumatic epilepsy, or in experiment, if we stimulate the cortex by faradic currents. The question arises, how the cortical impulse reaches the subcortical

centers of the tonus innervation. We know by the work of Economo and Karplus [19] in cats that after bilateral destruction not only of the pyramidal tract but also of the Pes pedunculi it is possible to get typical tonic and clonic fits following cortical stimulation. In other words, the Tract. temporopontin. and the Tract. frontopontin. are also not necessary to excite the lower centers from the cortex; only in apes, the pyramidal tract is more important for the conduction of cortical impulses not only for normal movements but also for clonisms. Hering [20] observed after cutting the pyramidal tract no more clonisms of the limbs but cloni of the muscles of the face and of the head.

We have to conclude that the cortical impulses reach the brainstem not only by the pyramidal tract but also by the extra-pyramidal system during epileptic seizures. We have here many possibilities for the conduction of the cortical impulse: for instance, the frontothalamic tract reaching the optic thalamus, from here fibers to the globus pallidus and from this ganglion pallidofugal fibers to deeper centers.

To analyze the relation between cortex and subcortical centers, the study of the *genesis of generalized epileptic fits* seems a good way. You know from clinical experience that such a seizure starts often in one limb, that the other muscles of the same side follow corresponding to the arrangement of the cortical centers and that finally also the muscles of the opposite side of the body show the tonic and clonic spasms. The same is true for animal experiments. If we stimulate, for instance, the left motor area, we get, first tonic and clonic fits of the muscles of the right side, finally also, in the hind leg and in the fore limb of the same side. Lewandowsky [21] and others believed that the corpus callosum is the way by which the impulse starting, for instance, in the left cortex, reaches the motor area of the opposite side. But experiments of Unverricht,[22] Franck and Pitres,[23] and in the last years of Karplus,[24] who made a careful anatomical control, showed that even after cutting the corpus callosum, a local stimulation of the cortex may produce, in dogs and apes, general epileptic convulsions. Karplus suggests the possibility that the impulse starting in the motor area of one side, reaches the opposite side by taking its way through the brainstem; he showed by hemisection of the spinal cord, that the cortical impulse has to reach the opposite side cranial from the cord but he made no experi-

ments to analyze the importance of the other commissural fibres of the forebrain or to show in which part of the brainstem the cortical impulse may cross the midline.

One may suggest, as also Karplus did, that changes in the excitation of the vasomotor centers play a rôle in the genesis of the generalized fit. We know by many clinical observations during operations (Krause,[35] Marburg and Ranzi,[36] Tilmann [37]) that the brain is swelling during the epileptic fit. This increase of intracranial pressure is the consequence of the convulsions of the skeletal muscles. Tilmann, for instance, who had put a manometer into the ventricle, observed the beginning of the increase of pressure some seconds after the beginning of the first contractions of skeletal muscles. But one could suggest that changes of the innervation of the vasomotors or of the intra-cranial pressure, play some rôle for the genesis, not of the fit itself, but for the generalization of the seizure. It could be possible, for instance, that the impulse, starting from the cortex, reaches the subcortical vasomotor centers and changes by this way the circulation of the brain, so that the spasm of skeletal muscles which was first local, becomes by the change of the blood supply of the brain or of the intra-cranial pressure, a general seizure. To analyze these questions, we made experiments in collaboration with Falkiewicz ; [38] we stimulated in dogs the motor area, for instance, of the left side, and put above the brain of the opposite side a plethysmograph to study the changes of the vessels of the brain or, if the dura was closed, the changes of the intracranial pressure. These experiments showed that after a localized cortical stimulation, a general seizure may be produced before considerable changes of the brain plethysmogram are observed. The generalization of the convulsions develops independently from the changes of the brain circulation or of the intra-cranial pressure, also if the skull is largely opened. Thus, we may say, that the excitation of vasomotor centers cannot be the cause of the generalization of the convulsions.

The question arises, how the cortical impulse starting, for instance, in the left motor area, reaches the opposite cortex. To get a true epileptiform seizure, which shows not only tonic but also clonic convulsions, it is indeed necessary that the motor area of the opposite side is reached by the cortical impulse. It has been shown by Karplus, who extirpated the motor area of one side and stimu-

lated the motor cortex of the opposite side; he found tonic and clonic convulsions only on the limbs homo-lateral to the side of the extirpation and on the opposite side only tonic convulsions. (Compare also former observations of Bubnoff and Heidenhain.[29]) We made similar observations: in one of our dogs, for instance, the cortical center of the fore leg and of the hind leg was excitable on the left side, but on the right side only the center of the fore leg was excitable, the center of the hind leg had lost its excitability, probably by mechanical injury. If we stimulated the motor area of the left side, we observed tonic and clonic convulsions of the right sided limbs and of the left sided fore limb, but no clonism of the left sided hind leg.

The former researches had shown that the corpus callosum is not necessary for the generalization of the cortical impulse during an epileptic fit which is produced by cortical stimulation. The question arises, if the other commissural or crossing fibres of the forebrain play some rôle. We cut, in dogs, in collaboration with Falkiewicz,[28] by sagittal sections, all fibres which cross the midline cranial from the rhombencephalon, in other words, the fibres crossing in the forebrain, in the diencephalon and in the midbrain (all experiments were anatomically examined). Even after this operation, we could observe that general clonic convulsions developed following one-sided cortical stimulation. These general convulsions were not the consequence of the spread of the stimulating faradic current, as they developed in some experiments some time after the electric stimulation had stopped.

We must conclude that the fibres which cross the mid-line cranial from the rhombencephalon are not necessary for the development of generalized epileptic fits. After these fibres had been cut, the general clonisms developed in the following way. The cortical impulse, for instance from the left motor area, had to descend to the pons and had to cross the mid-line in the rhombencephalon; from here it was conducted to the cortex of the opposite side. Such a conduction is possible even after cutting the decussation of the brachium conjunctivum by sagittal section of the midbrain. In other words, the conduction of these impulses from one cortex to the cortex of the opposite side is possible by extracerebellar tracts. We have no exact knowledge of the details of this conduction. We can only suggest some possibilities. We know for instance by the

experiments of Spiegel and Teschler [30] that the impulses from the cortical centers for conjugate movements of the eyes in the frontal lobe reach the vestibular nuclei. These nuclei send fibres to the subst. reticularis of the same and of the opposite side and from here following the investigations of Held to the optic thalamus; the impulse may finally reach the cortex by thalamocortical tracts.

It has been shown that the cortical impulse reaches deeper subcortical centers and especially the rhombencephalon and that on the other side the subcortical ganglia may have themselves an influence on the cortex. The question arises, if this mechanism is the only way of conduction of the cortical impulses during the generalization of an epileptic seizure or if the fibres which cross the mid-line cranial from the rhombencephalon play also some rôle in this mechanism.

To answer this question we made experiments in which we made a *sagittal section of the rhombencephalon in the mid-line.* The acute experiments gave no definite results. It was necessary to repeat these experiments in animals which lived some time after such a sagittal section of the rhombencephalon. In collaboration with Takagi [31] we opened in dogs the Membrana atlanto-occipitalis posterior and made the sagittal section in the median line of the rhombencephalon as slowly as possible to get as little trouble of respiration as possible. In the animals which lived some days after this operation the motor area was exposed on both sides and now the cortex on the left or on the right side stimulated. In these experiments it was possible to show that even after the sagittal section of the rhombencephalon clonic seizures could develop following stimulation of the cortex of one side on the limbs of the opposite and also of the same side. All possible precautions were naturally always made to avoid a spreading of the faradic current from the side of stimulation to the opposite side. The clonic seizures which we observed in these experiments were weaker on the limbs of the side of the stimulation than of the opposite side, but they were surely not the consequence of a simple spreading of the faradic current as they developed sometimes also some seconds after the stimulation had stopped.

If we compare these observations with the experiments of Spiegel and Falkiewicz we must conclude that there exist several ways for the conduction of the cortical impulse from one side to

the motor area of the opposite side. The last mentioned experiments had shown that the fibres which cross the mid-line in the forebrain, in the diencephalon and in the midbrain are not necessary for the development of generalized epileptic fits and that the cortical impulse may reach the opposite motor area by the help of crossing fibres of the rhombencephalon. The experiments in collaboration with Takagi show that after the sagittal section of the rhombencephalon generalized epileptic fits may also develop. In other words under normal conditions generalized epileptic fits may develop by the use of two groups of fibres, either of the fibres which cross the mid-line in the rhombencephalon or the fibres which cross it cranial from the pons. We know that pathological symptoms are produced only by changes of mechanisms which are normally pre-existing. Thus these experiments may show how many possibilities exist in the central nervous system for the conduction of cortical impulses.

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DISCUSSION.

Dr. F. H. Pike (Columbia University, New York City).—To discuss a paper like this on the spur of the moment after hearing it once, I think, leads one into temptation to say things which he might not say on more careful study of the data. There is little doubt, I think, that something has happened to the nerve cells in a patient subject to epilepsy which has affected their resistance to stimulating agents. If we are to get an explanation in terms of causes, we must show exactly what those conditions are. That I believe has not yet been done.

On the matter of anatomical spread, the rôle of the pyramidal tract and others, I think there has been a great deal of confusion. Some years ago we had introduced in physiology the conception of inhibition. It arose at a time when there was very little comparative data on the structure of the nervous system and it is based on the supposition that there has been immutability of species in the nervous system so far as function is concerned. That may seem surprising to some of you, but if you will go back to Setschenow's paper in the sixties and Goltz's paper in the seventies, you will find exactly that basis. The methods that we have used in the production of convulsions have been mostly chemical, whereas Dr. Spiegel has used electrical excitation. His problem is somewhat different from ours. Ours has been to find out these cells from which, under normal conditions, the impulses leading to the tonic and the clonic manifestations arise.

Dr. Spiegel has used electrical excitation and his problem has been to find the pathways. There may be some common elements—I think there are some common elements in the two problems.

The idea of tone in muscles is one which I may take up a little later in a paper which I will present this morning and perhaps at that time I can show some diagrams which may bear on this subject of spread.

I might mention in the great possibilities of spread that I have now in my laboratory a cat in which I made a hemisection of the spinal cord in the

lower thoracic region many months ago. It has long been known that after a hemisection the cat and dog regain the use of the limb on the side of the hemisection. Some months after that, I cut the decussation of the rubrospinal tract in the midbrain, leaving the descending pyramidal tracts intact. There is nothing going down the cord on the left side, but cutting the decussation of the rubrospinal tracts again brought on a motor difficulty in the left hind leg. It would look as if the rubrospinal tract going down on the right side of the spinal cord may have something to do with recovery of function in that left limb.

The cat still walked, so some weeks later we removed the left motor area and again there was a reappearance of the motor difficulty in the left hind leg. The left hind leg is now more severely affected, of course, than the right. But how the pyramidal tract originating in the left motor area and crossing to the right side of the cord finally gets across to the neurons of the left hind leg is something which puzzled us. That is only one of the problems we have in tracing out possible pathways.

Dr. Adolf Meyer (Baltimore, Md.).—The problem of fits of epilepsy is one of those that make us either blush or get pale when we are actually confronted occasionally with the attack, as I was two days ago when my daughter of fourteen came to me with an orphaned rabbit she was trying to raise, but had found in convulsions, urging me to do something about it. All I could say was that something had happened which started a condition I was unable to interrupt. We might possibly have to say we deal with a normal reaction under abnormal conditions. We then want to study the detail of so potentially a normal reaction of the nervous system to abnormal conditions. We can do it either by making a study of the conditions irrespective of the details of the nervous system or realize that we make largely a study of the details of the nervous system.

The points that have been brought out seem to be of this latter type; they amplify to a very large extent our knowledge of the normal functioning of the nervous system under important abnormal conditions produced in the status of the nervous system itself. What has been discussed both by Dr. Spiegel and by Dr. Pike leads us immediately into the details of working of the nervous system and such aspects as the permeability problem or the spreading problem, and we come with our knowledge of membranes and with our knowledge of various tracts and we want to see how those various items behave in attacks.

We are undoubtedly there profiting from the impetus to study one of the most diffuse and formidable reactions to the advantage of more knowledge of details and we are indeed led towards valuable knowledge of the internal working. For the purpose of determining what the march of the process of an epileptic convulsion is, we try to add additional difficulties to the "normal functioning under abnormal conditions," and it is very interesting to see how the experiments of Professor Spiegel show up a good many of our views of the functioning of the nervous system as probably too anthropocentric. We have got to recognize that a great deal of functioning takes place

in what I usually speak of as the segmental nervous system, the reflex levels and the inter-connection of the reflex parts, and that the working of the cortex is not quite as exclusively essential and responsible for all the detail of the fit as one usually thinks when one sees in epilepsy practically only a disequilibrium of the status of the hemispheres. Evidently the separation of the two halves of the brain perhaps does not so much involve specially responsible tracts as it shows that, once started from the one side or the other, the segmental nervous system will get into that status of osmotic changes, whatever they may be, in which an all-or-none function starts, a sweeping reaction, and this reaction *may* be partly checked by destroying one part or the other of one side. But after all, it is not so much individual tracts, the red nucleus, or, when we speak of extrapyramidal connections, the efferent paths minus the pyramidal tract, that do what we see; we had better say that in spite of the absence of the cortex, in spite of the absence of the red nucleus, we get such and such reactions.

Cutting the brain in the middle leaves after all a good many parts of the nervous system within the possibilities of operating. In other words, we evidently have not completely destroyed the possibility of "normal reaction to abnormal conditions." The normal reaction evidently is fundamentally dependent on the segmental apparatus that is still connected with the superstructures; by cutting the median line, we do not cause as much of an interference as we might usually think. I might just recall the fact that Starlinger and others have shown that cutting the pyramidal tract in its decussation leaves the animal so that you would practically not be able to say that anything had happened.

So I would just sum up by saying that the problem of epilepsy, the pathology of epilepsy, is partly a study of external conditions that bring about in the nervous system a potentially normal reaction to a special condition; on the other hand, we can study within the nervous system the rôle of the participant parts. There is hope that if we know more of what happens to the nervous system, we shall be able to do something. We are trying to do that by our hygiene, by a great deal of our medication, by lowering the reactivity of the nervous system and then we may possibly be able to get a great deal more light upon the nervous system and the ways it reacts under abnormal and normal conditions without, however, having come much nearer to the specific nature and causal conditions of epilepsy. I feel that we owe Dr. Spiegel our thanks for bringing before us something which makes us consider very definitely what we are doing in our experimentation in our epilepsy studies.

Dr. Ernest Spiegel (closing).—I thank you for your kind interest. I can answer only as far as I was able to hear these remarks. I quite agree with Dr. Pike that there may be sometimes different results if we make electrical stimulation of the cortex and if we make a general poisoning. I had some experiments with general poisoning, but I had my reasons why I didn't use for the question of the genesis of generalized fits such general poisoning.

If one, for instance, studies the reaction of the subcortical centers for general poisoning after cortical destruction and one finds clonism in such animals after absinth poisoning like in Dr. Pike's experiments, it shows that the subcortical centers may react by clonism and that there exist possibilities that clonism may have the origin in subcortical parts. But it doesn't show that clonism is of subcortical origin under normal conditions, if the cortex is in its normal connections with the subcortical centers. If we destroy the cortex and wait some weeks, then there develop what Munk has called the isolation phenomena. It means that the subcortical centers and the spinal cord get by the consequence of isolation new qualities, or qualities that they had not normally; one can find not only changes of the reflexes but also of the reactions following intoxication; the observation of clonisms in decorticated animals following absinth poisoning is therefore no sure proof that the clonisms are not of cortical origin if the cortex is intact. For the study of the genesis of the generalized epileptic fits, I thought it would be better to make localized faradic stimulation of the motor area, as these clonisms are surely of cortical origin. If one extirpates for instance in an animal the motor area of the cortex on one side, on the right side for instance, and stimulates the motor area on the left side, then one gets clonism only on the right side and never on the left sided limbs. It shows that generalized clonisms develop only in this case if the impulse coming from the left cortex reaches the cortex of the opposite side.

The experiment of a hemisection mentioned by Professor Pike is rather interesting. I may mention here that Karplus some years ago made experiments with hemisection of the cord to study the question of the generalization of fits. He showed that this generalization is due to supraspinal mechanisms. Dr. Pike mentions his experiments in connection with the rubrospinal tract. We made some experiments some years ago with an Hungarian doctor, Dr. Koernyey, in which we cut the midbrain following Sherrington's method and stimulated the surface of the transversal section. Graham Brown, as you know, made similar experiments and he found changes of the posture of the limbs. We could confirm this observation and found furthermore change of the tonus of the trunk muscles. Graham Brown believed that these reactions are a consequence of stimulation of the rubrospinal tract. In our experiments we made a sagittal section of the midbrain and cut both rubrospinal tracts and even then after cutting the rubrospinal tract by sagittal section, we got such reactions of the limbs and the trunk following the stimulation of the midbrain. There must be other tracts outside the rubrospinal tract which carry the impulses; also the pyramidal tract, the fasc. longitudin. post. and the thalamoolivary tract are not necessary for these reactions. It seems that systems of the substantia nigra play here a rôle, and I quite agree with Dr. Pike that the conduction of centrifugal impulses is a question that is very complicated.

When I mentioned the extrapyramidal tracts I meant all centrifugal fibers lying outside of the pyramidal tract, not only the striatum-pallidum system. The comparison Professor Meyer made between the observations of epileptic

fits in animals with severance of the corticofugal tracts and the experiments made by Starlinger, Hering, Rothmann and others who studied the movements of animals after cutting these tracts is very interesting. A similar comparison is also interesting concerning the corpus callosum. I mentioned the fact that if you make a sagittal section of the corpus callosum, even then we get a generalized fit and on the other side Trendelenburg made a section of the corpus callosum in apes and showed that in these monkeys even there was no marked trouble of movements. Thus it has been shown from the normal side like from the pathological side in our experiments how many possibilities of conduction of impulses exist here.

CEREBRAL TRAUMA AND ITS RELATION TO MENTAL DEFICIENCY.*

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To explain all cases of mental deficiency on the basis of one type of lesion is impossible and this is not my purpose. Two great groups may be recognized and separated immediately from the types of cases we are discussing: (1) Malformations of the central nervous system; (2) intrauterine brain diseases. In the first group one need only mention that tuberose sclerosis has been found in some cases as an organic explanation for the clinical picture. It is to be stressed here that at times only microscopic evidence of this condition has been found, in proportion to the diligence of the search. In looking through hundreds of sections from cases of mental deficiency, with or without convulsive manifestations, one is ofttime rewarded by finding occasionally the characteristic ectopic, large, atypical ganglion cells as evidence of this condition and an explanation for the abnormality of mental development.

It has also been shown fairly conclusively that intra-uterine encephalitis may occur.[1] This is, of course, sufficient to explain the slow mental progress or even lack of progress in cases of this sort. While probably not a frequent condition it should, nevertheless, be kept in mind in the study of cases of so-called mental deficiency. Syphilis must also be considered in any child with an inborn defect.

Abnormalities of development and diseases are many and varied and are sufficient cause for mental defects. It is, therefore, to be understood that the cases we have selected and are describing, are those in which there were no gross defects of these types, and no abnormalities of development. It is in this kind of case that the

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brain should mature normally and the child show no mental defects, It is in this type of case that a birth trauma may be the means of producing mental deficiency and a convulsive state.

It is now well established that subarachnoid hemorrhage is a not infrequent finding in the new-born. That it can and does occur without severe difficulties attending birth is well known. That it is not the entire cause of the fully developed cerebral diplegia or Little's disease is probably also true, but that it does have a relation to convulsive attacks and mental deterioration is within the realms of possibility. It is to the latter phase that this contribution is dedicated.

Frequency of Hemorrhage in the Newborn.

Cruveilhier noted, more than a century ago, that meningeal hemorrhage was not uncommon after difficult labor. Little mentioned it in his monograph of 1897 only to dismiss it as a cause of his disease. Weyhe, in 957 autopsies upon children from a few days to six months old, found signs of meningeal hemorrhage in 122 of the cases or in 13 per cent. Since this time the relative frequency of meningeal hemorrhage in the newborn has been verified by many observers, chiefly by Sharpe[8] of New York, who in the routine performance of spinal punctures in the newborn, has found free blood in 8 to 13 per cent of his cases. It is undoubtedly true that in a great many cases this blood is small in amount and very easily taken care of in the subarachnoid space, but in some there is such an amount that it provokes a reaction on the part of the soft meninges and the cortex. It might be interesting at this juncture to examine in detail just what occurs when red cells become free in the subarachnoid space.

Effect of Free Blood in the Subarachnoid Space.

(*a*) *Changes in the Pia.*—It is well known that the introduction of free sterile blood into the subarachnoid space sets up what has been called an aseptic meningitis. While there may be temporarily an outpouring of polynuclear cells and later lymphocytes, there is shortly the appearance of large phagocytic cells, the macrophages, (Figs. 1 and 2). One can see in these illustrations the large phagocytic elements filled with blood pigment lying within the subarach-

noid meshwork. At the same time there is a proliferation of the fixed tissue elements, the fibroblasts. This occurs even early, and even more so as the condition becomes more chronic, and especially when the blood is not drained off. So great may this fibroblastic proliferation be that there may result an obliteration of the free spaces within the cerebro-spinal fluid reservoir. If this occurs in the pathway of the fluid drainage towards the arachnoid villi at the vertex, blockage occurs, with resultant secondary or communicating hydrocephalus (Dandy,[2] Globus [3]).

A case illustrating this has recently come to my attention where as the result of a severe trauma, in a child, there occurred a hemorrhage into the subarachnoid space around the base of the brain. There soon occurred all the signs of symptoms of a progressive increase of intracranial pressure which was believed to be due to a brain tumor. A decompressive operation was performed without relief. At death, many months later, the true nature of the condition was revealed: complete obliteration of the spinal fluid spaces on the inferior surface of the frontal lobes and the cisterna chiasmatis, with back pressure, resulting in a severe internal hydrocephalus.

(*b*) *Changes in the Cortex.*—It has come to my attention in the pathological studies of the various forms of meningeal inflammations that the cortex does not remain passive. Changes of two types occur. In the first place as a result of the continuous irritation one sees in all forms of meningitis, and the meningeal irritation due to blood is no exception, that the marginal glial layer shows proliferative activity. One sees very shortly a swelling and proliferation of the marginal glia. The Cagal stain brings this out vividly but even in the toluidin blue stains it is to be seen. One notes in Fig. 1 the prominence of the marginal glial layer, brought out even in the toluidin blue preparation. This is the type of defense reaction on the part of the cortex from all other forms of subarachnoid irritation. This marginal gliosis has been considered characteristic of "epilepsy" but it is probably only evidence of irritation within the subarachnoid space.

The second result from the irritation and fibrosis in the soft meninges is the more or less marked interference with the blood supply to the cortex so that the ganglionic elements are deprived of their full nutriment and mild or even severe changes can result.

The generalized gliosis of the macroglial type is a good index of the nutritional disturbance and that is what occurs. This nutritional disturbance is usually not severe enough to lead to actual necrosis of tissue with gitter cell formation so that one looking for gross damage to the cortex may overlook the relatively mild changes that occur. It is this same type of change that is to be noted in the brain of an adult, the result of general arteriosclerosis, where gross lesions are absent, but in looking over the cortex one sees a generalized poverty of ganglion cells, while those that remain show mild or even severe degenerative changes. It is because of the mildness of the changes that the pathologist who is only trained to pick out gross lesions finds but little wrong with the cortex in these cases. This type of change in the ganglionic elements is illustrated in Fig. 1, where there has been only a marked decrease in the number of ganglion cells but those that remain show degenerative changes of all sorts, mainly of the so-called ischemic type.

Hindrance to Development of the Pacchionian System.

It has been shown by Globus,[3] particularly, that in some infants brains the soft meninges may retain their embryonic character, *i. e.*, they show no subarachnoid space. While a developmental fault can account for this it is just as likely to result from the irritation of free blood in the subarachnoid space from birth trauma even though Globus himself found no inflammatory elements present at the time of his examination. As a result of the sealing up of the subarachnoid space, especially over the vertex the normal development of the arachnoid villi and Pacchionian bodies is hindered. Thus, we have found an aplasia of these structures in a small percentage of cases, and in others a poor development of these eliminative structures.

Another phase of the same problem, which, theoretically, should not be discussed under the designation of cerebral trauma, is, nevertheless, so closely related that it can, with benefit, be taken up here. That is the question of cerebral anemia or anoxemia. It occurs in the newborn when breathing is delayed for any abnormal interval. We have learned from experimental work that the absence of oxygen produces certain changes in the brain tissue in spite of the apparent contrary findings of Ford[4] who felt that trauma is more

important than anoxemia. Gildea and Cobb [5] are inclined to disbelieve this and have shown that definite changes occur with shutting down of the circulation, and they conclude that "ten minutes of cerebral anemia is sufficient to impair cortical cells permanently" (p. 901). In infants who fail to breathe at the time of birth and therefore die, the changes in the brain are not "fixed" long enough to enable us to study the changes under the microscope by the staining methods now in use. It was, therefore, extremely fortunate that I obtained the brain of a patient, an adult, who stopped breathing for a little over five minutes during the course of a minor operation, and then was resuscitated and lived for five days. Here the changes were so well established that they gave us an inkling of what happens in the newborn under similar conditions. In this brain there was such extensive degeneration of brain tissue that one does not wonder if there is a mental deficit in a child under these conditions. This case will be described in full in the literature. Suffice it to say here that the changes were noted in the lining cells of the small capillaries which were so swollen as to obstruct the lumina of the vessels. Landis [7] has shown experimentally that when oxygen is absent, fluid enters the tissues at four times the usual speed and this may have a great bearing on the problem. Numerous areas of rarefaction (verödungsherde) were interspersed in the generalized degeneration taking place throughout the entire central nervous system. No one could doubt the mental changes that would occur in an individual with these changes, were he unfortunate enough to survive. These cases may not have the clinical syndrome of cerebral diplegia, but show an amentia, in proportion to the cortical damage.

If, however, the changes in the meninges do not occur at birth but the fluid pathway is well established before bleeding into the subarachnoid space occurs, then the changes in the arachnoidal system cause a damming back of the fluid. This is the type of change seen so exquisitely in the following case of the late infantile form of Tay-Sachs disease: M. B., a girl of 4, whose family history showed no abnormalities, was admitted to my service 11-5-1929, because of failure of development. There has been inability not only to walk and talk, but the child was unable to sit up or support its head.

On examination one noted a child who was physically well developed. The head was relatively large and the sutures wide. Nystagmus was present. The pupils were equal and regular and reacted sluggishly. The child appeared to be blind. It was very restless and the reflexes difficult to elicit. Babinski reflex was present on both sides. Athetoid movements were present in all limbs. Hyperacusis could be demonstrated by a sudden noise which would almost throw the child into a general convulsive movement. While in the hospital the child had a generalized convulsion.

Eye examination showed that the case was in all probability one of amaurotic family idiocy, with atrophy of the optic nerves, not of the primary variety. Both maculæ showed a rather brilliant white centre but they merged with the surrounding retina which was more or less granular.

An encephalogram was done by Dr. Fay, with the result shown in Figs. 3 and 4, of marked internal and external hydrocephalus.

In cases of this sort one suspects that the usual fluid pathways had been established and that back-pressure had gradually resulted in the atrophy of the cortex, in the fluid field, the fronto-parietal area as result of pressure-atrophy (Fig. 4). The old theory that cortical atrophy is the result of the underlying cortical and subcortical degeneration no longer holds, has been shown by Fay and Winkelman.[8] This has recently been impressed upon us by a case of Schilder's disease, in the brain of which there was the usual severe subcortical degeneration, particularly in the occipital and temporal areas with practically complete absence of subcortical damage in the frontal and parietal areas. Yet in this case the superficial cortical atrophy was intense in the fronto-parietal areas and practically absent in the temporo-occipital areas. This case by no means represents an isolated instance of this state of affairs.

Mental Deterioration in Adults After Cerebral Trauma.

There is a large group of adolescent and adult cases in whom mental deterioration results from cerebral trauma. These patients shortly after the trauma complain of dizziness and headache. Later there are changes in disposition and character with irritability, lack of ambition, restlessness and vague complaints that are all too frequently classified under the heading of a "traumatic neurosis."

Convulsive manifestations are unfortunately common. From the Foerster clinic in Breslau, there have been reported the results of the encephalographic findings of gross and visible lesions in these traumatic cases. Following their lead we have done many encephalograms in the post-traumatic cases and have found two great groups of cases: (1) Those with gross focal lesions. Here one sees unilateral dilatation of one part of the ventricle with deviation towards the injured side, as result of contraction of the scar tissue. Penfield has gone into the mechanism of the histology of these scars from the experimental angle, and has recently made suggestions as to therapeusis. This group of cases does not concern us at present. (2) Encephalography has disclosed a characteristic atrophy of the brain following cerebral injuries. The complete drainage of the spinal fluid system and its replacement by air has revealed that these cases show characteristic shrinkage of the frontal lobe, the islands of Reil and the motor and parietal areas of the brain. The striking loss of brain volume is difficult to determine in any other way, as frequently following removal of the brain from the skull one loses the relationship in volume in comparison with the cranial cavity and hence only an approximate idea of the volume of brain loss can be ascertained. Encephalography makes possible the viewing of the brain by the stereoscopic method almost as though the structure were placed in the hands for inspection. The cortical patterns and actual relationships between the ventricular and subarachnoid spaces can thus be determined with great accuracy. The method further offers a means of determining the loss of brain volume in a fairly definite manner by the amount of spinal fluid obtained at the time of the complete drainage. The normal amount of fluid obtained in the adult whose skull is not deformed or unusual in size approximates 110 to 130 cc. Many cases in which brain atrophy follows cerebral trauma yield between 140 to 280 cc. of fluid, although the external cranial markings and size give no indication of this apparent hydrocephalus. Encephalographic findings in infancy and childhood indicate that the brain suffers a rapid diminution in volume and shrinkage in the fronto-parietal areas, following birth trauma, and subarachnoid hemorrhage which is frequently spontaneously present and unsuspected in normal deliveries. The changes which may be found within three weeks to six months are strikingly similar to those in

later periods of life. The process occurs rapidly and it is our belief that they are similar to secondary pressure, especially hydraulic pressure, over the fronto-parietal cerebrospinal fluid pathways.

There results a mechanical pressure in those areas of the brain over which the fluid must flow to reach its point of exit, the arachnoid villi. Increase in pressure, the result of defective elimination, registers directly upon these fluid pathways. Defective elimination can result from impairment of the filters along the longitudinal sinus from inflammation, edema, fibrosis, or hemorrhagic infiltration. The result of this long continued mechanical pressure on the fronto-parietal cortex by what is the closest fitting cast that can be applied to the cortex—an hydraulic cast—produces very shortly a definite atrophy as shown by widening of the sulci. This is recognized in the encephalogram by the increase in width of the air pockets over the cortex. There may be another factor concerned, and that is the concentration of the metabolic products from the brain substance which have been emptied into the subarachnoid space. One sees many times a reaction produced in the meninges, leading to fibrosis, in cases in which breaking down of brain substance has occurred. In case of defective elimination of these waste substances from the subarachnoid space they are concentrated in the region of the Pacchionian bodies, and it is here that the most intense fibrosis occurs in the meninges. One must not also forget the gliosis that occurs in the cortex itself from irritation within the subarachnoid space. There may thus be established a vicious circle.

Our own work on cortical atrophy (Fay and Winkelman[8]) has demonstrated to us rather conclusively several facts: First, that the cortical atrophy is greatest in the fronto-parietal areas of the brain. Secondly, that this represents the fluid field of the cortex, and, thirdly, that the atrophy is probably the result of pressure from overlying fluid. It is exactly the same type of atrophic changes that occurs from an overlying slowly growing tumor or slowly expanding blood clot, except that the fluid is a much more snugly fitting covering and does not obliterate the convolutional markings but rather exaggerates them.

The effect on the brain of trauma whether at the time of birth or afterwards can be so marked as to produce mental deterioration of rather severe grade, as well as convulsive attacks. With the

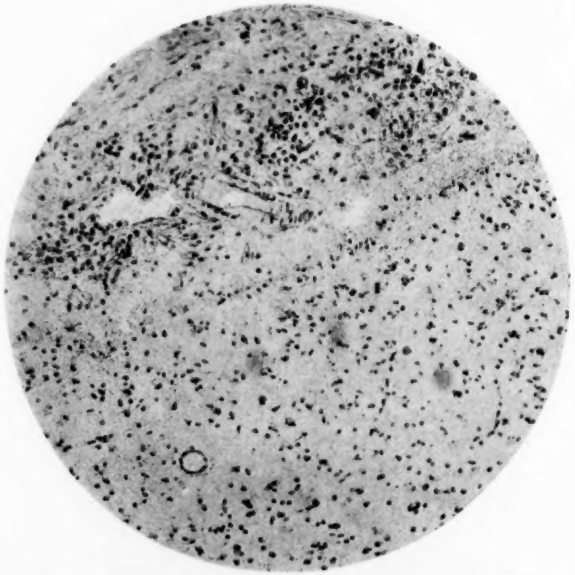

FIG. 1.—Toluidin blue stain. Subarachnoid space filled with pigment-laden phagocytic cells (macrophages). A marginal gliosis is present. Severe ganglionic degenerative changes can also be made out universally throughout the cortical area.

FIG. 2.—Toluidin blue stain. Pigment-laden phagocytes present within the subarachnoid space. An area of rarefaction (verödungsherd) seen in cortex, with beginning glial proliferation.

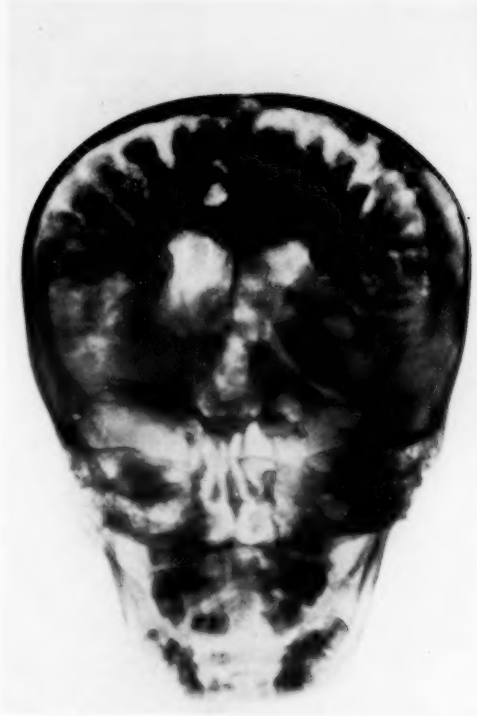

FIG. 3.—Encephalogram showing intense external hydrocephalus, with moderate ventricular dilatation.

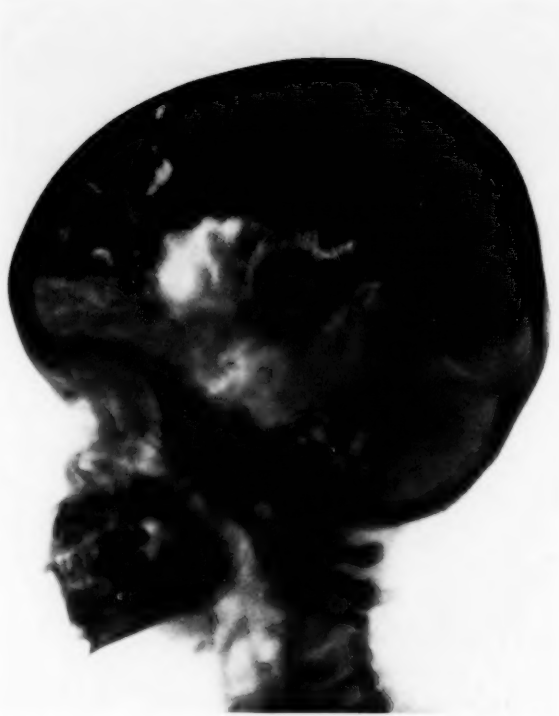

FIG. 4.—Encephalogram showing atrophy in fluid field (fronto-parietal area).

limitation of the spinal fluid excess by means of strict dehydration many of these cases have shown prompt improvement in mentality, alertness and progressive development of speech and motor faculties. The subsequent development of the child with such a handicap depends upon the amount of cerebral tissue remaining. It has been striking to see that a fairly satisfactory degree of mentality can be promoted even with large and gross cerebral defects. Memory is usually acute for visual impressions, as well as auditory, as this characteristic hydraulic pressure atrophy does not affect the temporal or occipital lobes. It is extremely important to note that these faculties remain intact while those such as judgment, behavior, control and inhibition as well as psychomotor activities seem to suffer most.

Conclusions.

1. Cerebral trauma plays a part in the deteriorations of adults and "arrested cerebral development" of infants.

2. Subarachnoid bleeding calls for repeated spinal drainage in order to lessen the after results.

3. Encephalography gives us visual proof of the post-traumatic atrophies present mainly in the fluid field, the fronto-parietal area.

4. Dehydration offers a means of improving the mental condition of both the infantile and adult traumatic cases.

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DISCUSSION.

Dr. Adolf Meyer (Baltimore, Md.)—I am sorry that I was not present during the whole demonstration, but I am very glad indeed to hear Dr. Winkelman put the emphasis he does on the importance of paying attention

to the blood in the cerebrospinal fluid. It was Dr. Charles Bagley who in our laboratory demonstrated with experimental material the importance of what Dr. Winkelman just mentioned and who has also shown in his practice in the treatment of infants with blood in the cerebrospinal fluid the importance of consistent and thorough drainage until the cerebrospinal fluid has rid itself of a component which evidently is objectionable.

DR. MORGAN B. HODSKINS (Palmer, Mass.).—Mr. Chairman, if it is true that the drainage of cerebrospinal fluid is through the Pacchionian granulations and disease of the Pacchionian granulations will cause a backing of the cerebrospinal fluid, thereby causing atrophy of the cerebral cortex, why doesn't the backing up dilate the ventricles first and cause hydrocephalus? I would like to know how Dandy's experiments can be explained when he says that the Pacchionian granulations have nothing to do with the drainage of the cerebrospinal fluid.

DR. N. W. WINKELMAN.—We have published, in 1929, a complete account of our findings in answer to the question. There is at the present time a discussion at Hopkins between Dandy and Weed on the mechanism of elimination of cerebrospinal fluid. I think Weed has shown very conclusively that the interpretation which he has placed upon the Pacchionian bodies is correct, substantiating the work of Key and Retzius many years before in 1876. Dandy has recently published a paper on what he calls "Communicating Hydrocephalus," where as the result of obstruction at the base of the brain there is internal hydrocephalus. He explains it a little differently than we do. We too have found an internal hydrocephalus where there is obstruction at the base, where, for example, in a tuberculous meningitis which has a basal location, one gets very quickly an internal hydrocephalus, more so than when there is obstruction near the superior sinus and when there forms an hydraulic cast over the cortex. The best form of cast that the cortex can have is the fluid. We have compared the cortical areas in a case showing frontal atrophy, the areas of atrophy and the areas of non-atrophy. The change is as night is to day, the change is so great. One can pick out the sections without looking at the labels. The change that we have found is a pressure atrophy, the same sort of pressure atrophy that one gets from pressure as the result of an endothelial tumor over the cortex. Wo do not claim to be the first to put forth the theory of the drainage of cerebrospinal fluid through the Pacchionian bodies. That is the work of Key, Retzius and Weed. We feel we have substantiated it.

DR. CHAPPELL.—It was stated that the cessation of breathing caused this rather interesting lesion. How are we to be certain that the lesion didn't cause the cessation of breathing? That would seem to require a little explanation.

CHAIRMAN DIXON.—There may be one or two more who have questions in mind to ask Dr. Winkelman before we close the discussion and we will give opportunity for it, at least we will take advantage of the people coming in.

Are there any other questions or any other comments to be made upon Dr. Winkelman's paper now before we ask him to close it definitely? Doctor, will you answer Dr. Chappell?

DR. N. W. WINKELMAN (closing).—Again, we have a very interesting problem that has been taken up from many, many angles. The effect of anoxemia on the brain has been taken up before; this is not an isolated instance. It has been taken up particularly by the Spielmeyer school and there have been reported many cases of this kind in the literature. Anoxemia produces on the brain cells a change which can be reproduced experimentally as done recently by Gildea and Cobb. Landis from the University of Pennsylvania has shown in a very excellent research the effect of lessening of oxygen where fluid leaves the vessels more readily.

While the question as to which is first is an excellent one, have we put the cart before the horse? It is really a very excellent question, but many cases of the same sort have been reported of the same thing that leave no doubt that the anoxemia was the prime factor; it produced the brain changes; they were progressive and death ensued as the result of the brain pathology. So that while it might be improper to state that death resulted from the anoxemia, it did result from the changes that anoxemia had produced.

EPILEPSY AND ITS RATIONAL EXTRA-INSTITUTIONAL TREATMENT.*

By DOUGLAS A. THOM, M. D., Boston, Mass.

The late Sir William Gowers, in the introduction to his well-known book, "Epilepsy and Other Convulsive Diseases," published in 1881, states, "The convulsions which occur as a result of chronic brain disease may be divided into two classes, (1) Those which are the result of organic disease, such as can be recognized after death, (2) Those which are the expression of a condition of the brain which is not evidenced by any visible alteration," the only evidence of disease being the disordered function. He speaks of this latter group as "depending upon such conditions of the nerve centers as elude detection by methods of examination at present at our disposal."

Nearly 50 years later Lennox and Cobb, in the closing paragraph of their monograph, published in 1928, entitled, "Epilepsy," reach the conclusion that "there is no constant anatomical lesion in epilepsy and only a minority of patients with extensive cerebral pathology have fits." The same authors further state, "We are forced to postulate some unknown constitutional element," referring to the fundamental factors which account for the convulsive phenomena, and go on to say, "Moreover, our meagre knowledge and crude methods do not permit us to measure this tendency."

In speculating about the physio-chemical forces which may conceivably be fundamental, in rendering a particular individual susceptible to fits, whether they are hysterical, epileptic, eclamptic or traumatic, Lennox and Cobb conclude that, "Though this variability is presumably related to the subtle chemistry of the cell, its elucidation is for the future."

If one were not acquainted with the literature on the subject of epilepsy one might well conclude that little advance had been made during the past half century in our understanding of this baffling

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disease. We are still speaking of our limited knowledge and crude methods which permit abnormalities, both pathological and biochemical, to pass by undetected; yet there has been, and still is, a small army of hard-working, conscientious, well-trained men and women driven on by the time-honored curiosity of the scientist, held to the task by indomitable patience, who have been laying the foundation for future research, which cannot fail to bring ultimate results. At the moment, however, we are still in a state of darkness as to even the physiological mechanisms by which convulsions are produced.

Hughlings Jackson, in the early seventies (1873), suggested that the convulsion was due to cerebral irritation, and was ardently supported by his colleague, Gowers, in this hypothesis. Sargent, in England, and Rosett, in this country, have more recently put forth the theory that the fit was due to a release phenomena of the cerebral cortex, allowing the lower centers to operate uninhibited. A modification of the release theory is that termed the short circuit theory. It implies that localized cortical lesions may bring about deflection of a group of nerve impulses by interrupting the association fibers, while the explosive theory assumes that the convulsion is caused by suddenly affecting brain tissue *en masse,* through chemical changes which are not clearly understood at this time.

Whatever the mechanism may be, whether confined to one of the four theories outlined, or some other (or perchance a combination of two or more different mechanisms), time alone will reveal. It will be doubtful if we find that the same mechanism is constant in every individual case, or that it operates consistently in the same individual. This lack of consistency is made quite obvious by the observations of convulsive phenomena associated with head injuries sustained in the war, where only from 5 to 8 per cent of such cases had fits, and again by the recent report made by Dr. H. L. Parker (Rochester, Minnesota, Research Association in Nervous and Mental Diseases, December, 1929), which indicated that there was no consistency in symptoms, particularly convulsions, in relation to brain tumors. The same type of tumor with an identical location would be associated with convulsions in one case and not in the next.

Turning our attention to the pathological investigations on the subject of epilepsy we are confronted with the fact that although

interesting and spectacular pathological lesions are found associated with the chronic convulsive disorders, we are still without the slightest clue of what may be called a characteristic pathological finding for epilepsy. Such lesions as have been found associated with the disease have not been sufficiently constant to make it at all conclusive that they represent the determining pathological process. Then again, the fact remains that similar lesions have been found associated with other clinical entities where convulsions have not been present.

The question has always arisen in the mind of the pathologist whether the lesion found was the cause or the effect of the convulsion, and invariably we are thrown back upon the dictum of Gowers, who stated, "Epilepsy is a disease of tissue, not of structure, a disease of the gray matter, it has no uniform seat."

Therapeutic measures, particularly those of a specific nature, have been equally disappointing. The earliest records of medical history bear witness to the fact that epilepsy has been attacked therapeutically from every available angle. The medicine man and other fanatics have approached the subject with all the enthusiasm of the mystic. Bleeding, trephining, injections of snake venom, surgery to the point of almost complete evisceration, have been practiced with but comparatively little success. The most encouraging approach from a therapeutic point of view is that which has come from treating the patient rather than the disease. Psychotherapy and modern dietetics are examples of this approach. Years ago the medical profession took the stand that epilepsy was not a disease, that it was but a symptom or a symptom complex, and that it represented nothing more than an expression of some well-defined or perhaps rather obscure pathological condition, and that careful clinical examinations and intense research investigations would solve the problem.

This conception of epilepsy has been most stimulating and has directed our attention to a more careful and painstaking approach to the problem of the convulsive disorders. We are no longer satisfied with a casual physical and neurological examination but we have delved deeply, and we hope intelligently, into the mental life of the patient. We have utilized all the advances that have been made in modern laboratory technique in our efforts to determine if biochemistry has not something to contribute to our under-

standing of the underlying cause of this baffling condition. Psychology and psychopathology have been utilized and the whole mental life of the patient has been subjected to intensive scrutiny. In brief, all that is available in medicine has been brought to bear upon the solution of the problem. We are still confronted with a large army of individuals suffering from convulsive disorders whose pathogenesis eludes detection.

I have made this hasty review of the present status of our knowledge regarding the mechanisms, pathology, and therapeutics of epilepsy, not to make record of our failure to solve the problem relating to factors underlying this disease, or to strike a pessimistic note on the limitations of research. Much of great value has been forthcoming from the clinical studies and researches of such men as Gowers, Jackson, Southard, Rows, McCurdy, Clark, Rosett, Musken, Geylein, Cobb, Lennox and others—all too numerous to mention. Rather has this preliminary statement been made in order to justify the approach to the problem which I am suggesting, namely that the treatment should be directed to the individual rather than the disease in all those cases suffering from the chronic convulsive disorders where definite pathological causes have eluded detection.

Until recently practically all of our observations on the subject of epilepsy have been considered from the institutional point of view. That is, our clinical observations, our pathological investigations, and, to a very large extent, our experimental therapy, have been confined to patients whose symptoms have been of such severity and long duration that they have necessitated their institutional supervision. This group not only represent less than 3 per cent of all individuals suffering from chronic convulsive disorders but as a group they appear to be quite different in their physical and mental symptomatology. A study made of the patients at the Monson State Hospital, Palmer, Massachusetts, some years ago, revealed that 90 per cent of all the inmates of that institution were either intellectually deteriorated or mentally deficient. Only 25 of the entire group of over 1000 patients could be looked upon as being normal mentally. One out of every three could not read or write. Pathologically two out of every three cases which came to autopsy revealed gross brain lesions visible to the naked eye upon

superficial examination. A large per cent showed neurological lesions indicating brain damage. The average duration of the convulsions of the adult cases before entering the hospital was 13.6 years. The social, cultural, intellectual and economic level of our institutional cases is obviously lower than that of the general population at large.

The institutional material represents the end results of some pathological process which undoubtedly bears a direct relation to convulsive disorders. The devastation that is produced by the convulsions themselves cannot be ignored. These cases stand out in marked contrast to the great majority of patients that one sees in our out-patient clinics and in private practice.

A report entitled, " Defects Found in Drafted Men, 1920," from the office of the Surgeon General, presents certain statistical information which is of value in estimating the incidence of epilepsy in the general population. Prior to the World War these statistics indicate that epilepsy was the most frequent neuropsychiatric condition coming to the attention of recruiting officers, but the figures covering the period for the World War show that epilepsy was preceded numerically by four other neuropsychiatric conditions, and that it constituted 9.2 per cent of the entire neuropsychiatric problem. The local boards were without instruction prior to November, 1917, to reject applicants on account of epilepsy. After that date 5.15 per thousand were rejected as epileptics by these boards, the total number being 14,195 cases.

It appears from a document issued under the title of, " Medical Department of the U. S. Army in the World War, Vol. X," that 6388 cases of epilepsy were detected in service, 76.2 per cent of which had fits for a period over five years. The social and economic condition of the epileptics as a group were not particularly different from the general run of service men. Seventy-five and eight-tenths per cent were single as compared with 86.8 per cent of the non-epileptics. Economically there was little difference.

Epilepsy was recorded in a varied amount in the different states; for example, Vermont showed 12.7 per thousand, Maryland 7.9 per thousand, Massachusetts 5.9 per thousand, while Louisiana had 6.88 per thousand. It was stated in the same report that " two-thirds of one per cent of the men of military age from the agricultural areas of the north—that area occupied by the largest

portion of native whites—were found to be epileptic." This of course excludes all those cases that were under institutional care.

Notwithstanding the fact that these figures deal with a rather selected group of the population, it is fair to point out that this particular group would have a very large representation in industry, agriculture, or other occupational pursuits because of their age.

It has been estimated by various authorities that from two to four in every thousand of the adult population suffer from epilepsy. Dr. Edward A. Tracy, in a survey not yet completed, of the Boston school population,* states that approximately four children per thousand between the ages of five and sixteen are suffering from convulsions. This represents a large group of children who are at least attempting to carry on in spite of their handicap and who are not incapacitated for school attendance. This of course indicates, as previously stated, that but a relatively small per cent of the population suffering from epilepsy, either children or adults, are in institutions. It would be difficult indeed to estimate just what proportion of this non-institutionalized group are well adjusted to society, and whose industrial inefficiency has been but little impaired.

The general practitioner and the neuropsychiatrist in private practice find that but a relatively small per cent of their epileptic patients are deteriorated intellectually; psychotic symptoms are infrequent and neurological signs indicating organic disease are the exception. It is true that we do see the feeble-minded child who is having convulsions, but the convulsions are probably part and parcel of the same pathogenic condition which produced the feeble-mindedness. We are all familiar with those personality changes which so commonly are associated with epilepsy or any chronic disease of long standing; irritability, hypochondriacal ideas, depressions, self-centeredness, are certainly not confined to the epileptic, and we find many individuals who have suffered from epilepsy for years quite free from the so-called "epileptic personality."

A large part of this non-institutionalized group are carrying on either in school, the store, shop, factory, office, or whatever their vocation may be. Inquiry has revealed that several large industries

* Personal communication.

have had epileptics in their employ over a long period of time, and the occupational history of those individuals coming to our outpatient dispensaries or our clinics, indicates that they are in a very large measure industrially efficient.

A recent survey of the jails and houses of correction and prisons in the State of Massachusetts, was made in an effort to determine the frequency of epilepsy among the reformatory and prison population. Twelve of the fourteen county institutions, with an average daily population of 2504 reported that they have no epileptics at the present time. One of the state institutions * with an average daily population of 725, reports that among the present population there are five epileptics sentenced for the following reasons: violation of the auto laws, abandoning an infant, vagrancy and assault and battery with a dangerous weapon, drunkenness and larceny.

The Reformatory for Women, which has an average daily population of 277, reported they have had but 10 epileptics since May 1, 1918. The Bridgewater State Hospital, an institution giving hospital care to the insane criminals, the defective delinquents, and also a portion of the prison population which are sentenced for minor offences, and having a combined population of 1932, reports 30 epileptics among their criminal population. It is of interest to note that the offences which brought these epileptics under the custodial care of the state were very largely of minor nature, such as, drunkenness, contempt of court, vagrancy, exposing and keeping for sale intoxicating liquors, and cases transferred from almshouses. In only two of these cases was there any attempt on the part of the epileptic to do bodily harm to another person.

These combined groups represent a fair cross-section of the prison population of Massachusetts at the present time and indicate that the type of crime committed by the epileptic is not unlike that which one would expect from any individual so handicapped that competition in the open labor market was difficult. Larceny, drunkenness, vagrancy, and general irresponsibility are the common offences of the epileptic. Of the 6388 cases of epilepsy reported by the Medical Department of the U. S. Army in the World War only 53 came up before the Disciplinary Boards.

* Deer Island.

This survey does not indicate, as we have been prone to believe, that the atrocious crimes of violence are common among the epileptics.

THERAPY.

Therapy for non-institutional cases:

(1) Every effort should be made to determine by a careful clinical examination and the application of laboratory tests, X-ray examinations, and all other available methods, such physical, biochemical, and psychogenic factors, that may directly or indirectly cause, aggravate, or perpetuate, the convulsions, bearing in mind that such organic factors can only be eliminated insofar as it is compatible with the patient's general, physical and mental well-being.

(2) Discuss your findings frankly with the patient. The results of a time-consuming, ofttimes discomforting and expensive examination, from which he is seeking relief and to which he is pinning his hopes for the future, are of vital importance to him.

(3) With equal frankness discuss the patient's incapacity in relation to his future happiness and efficiency.

a. What the patient can reasonably expect from therapy, whether it be an operation, the administration of drugs, dietetic measures, or whatever else may be indicated.

b. What can be expected by proper adjustment of certain physiological conditions which are working out to the disadvantage of the patient, such as regulating life habits—sleep, diet, bowels, sex, exercise, work, social activities—stressing the fact that any one or more of these factors may play an important part in reducing or eliminating the convulsions.

(4) Psychogenic approach.

a. Eliminate insofar as possible, the mystery and feeling of impending danger that surrounds the patient suffering from epilepsy. This may be done by removing the social stigma that is attached to the disease, by minimizing the importance of an occasional convulsion, and a frank statement of the hereditary aspects of the disease.

b. Create optimism instead of pessimism wherever justified, which means in all cases where intellectual deterioration and psychotic symptoms are not present.

c. Help the patient understand that he himself has much to contribute to his own cure by making intelligent observations as to conditions and circumstances which are associated with his convulsions, and by following explicitly the directions of the physician.

d. Impress the patient with the fact that because you have been unable to find any physiological factors to account for his illness up to the moment, this does not mean that interest and further investigations will cease.

e. Discuss with the patient perfectly frankly some of the researches that are being carried out to determine the causes for convulsions, keeping in mind that the epileptic needs all the hope and courage which the circumstances justify.

(5) Education of the family.

a. The head of the family and those coming in immediate contact with the patient should be given all the information that is essential to their having an intelligent understanding of the disease, that they may adopt a more helpful attitude toward the patient, encouraging optimism rather than pessimism and assist in carrying out instructions with greater wisdom and making such observations on the patient as will be useful to the physician. The patient must be encouraged to do for himself and others all that conditions will permit. In brief, all those individuals coming in contact with the patient must help him build his life around normality instead of abnormality.

(6) The same type of education should be extended to the employer or those coming in immediate contact with the patient outside the home.

(7) Careful consideration should be given to the question of the selection of vocation, and the type of education which is most likely to work out to the patient's advantage. One aspect in the treatment of epilepsy that has been stressed by the psychoanalytic school and steadily neglected by most general practitioners and neuropsychiatrists has been that of psychogenic factors or situations acting as the precipitating cause of the convulsions. This neglect has been due, in part at least, to the early antagonisms toward psychoanalysis in general, and in part to the time-consuming technique involved in carrying out the treatment. There are, however, innumerable psychogenic factors operating very near the level of consciousness and these can be removed without the long

drawn out psychoanalytic approach. These emotional situations are of greatest importance as precipitating factors for the convulsions. Often symptoms of this type are not recognized as part of epilepsy but are looked upon as hysterical manifestations. However that may be, they do represent a chronic convulsive disorder and must be treated as such.

I think it would be generally agreed among both psychiatrists and neurologists that convulsions, as a manifestation of hysteria, are relatively uncommon—that is, in comparison with other physical symptoms of mental conflicts, such as disorders of the special senses, and sensory disturbances and amnesias—and that there is greater danger from the standpoint of diagnosis in diagnosing epilepsy as hysteria than in calling hysteria epilepsy.

Of 50 cases studied at the out-patient clinic of the U. S. Veterans' Bureau diagnosed as hysteria, 17 were having convulsions as a part of the clinical picture when first admitted to the clinic. During a period of careful observation and study lasting six years it was necessary to change the diagnosis in seven of these cases to epilepsy. Fifteen of these 17 patients diagnosed as hysteria are continuing to have convulsions. These 15 cases are no less incapacitated than those cases diagnosed as epilepsy, yet the characteristic clinical manifestations of epilepsy are absent, and those indicating psychogenic origin of the fits are present. Rarely have we found it necessary to change the diagnosis from epilepsy to hysteria.

This does not mean that convulsions of an epileptic character with all the motor and mental phenomena, so characteristic of that condition, may not be precipitated by emotional situations. It simply stresses the point that in the treatment of these cases psychogenic situations which are operating at the moment cannot be ignored. The following case exemplifies this point:

Mrs. C., born in Connecticut, 25 years of age, birth and development normal. As a child she was considered unusually bright. When 10 years of age she had her first convulsion, and attended the clinic at the Children's Hospital. The convulsions were infrequent, one in two or three months, and did not prevent her from continuing her education to the second year in high school, when she left on account of an attack of rheumatic fever. The convulsions usually occurred at night, but since her marriage three years ago, she had attacks at infrequent intervals during the day. These attacks were usually precipitated by heated arguments with her husband.

There was apparently no aura, consciousness was lost completely, she bit her tongue, but did not wet or soil her clothing. Spells lasted about ten minutes and, from the description, had none of the dramatic characteristics associated with hysterical attacks.

The home situation was very chaotic when patient first came to the hospital, as the patient's father had accused her husband of endeavoring to have her put away in an institution on account of the frequency of her convulsions. Patient's mother-in-law had her child and she was not permitted to see it, and the patient was living with her own mother and her husband was living at his home.

In spite of this estrangement, there seemed to be a desire on the part of both husband and wife for reconciliation and the case was turned over to the Social Service. The family difficulties were ironed out, and the patient and her husband and child separated from the other members of the family and started to build up a little home of their own. This was in October. Patient was seen last May 17. At that time she had not had a convulsion for eight months and things at home were going much more satisfactorily.

The point of interest in this case is the fact that this woman having convulsions, beginning at the age of 10, which were epileptic in character, should at the age of 22 begin having attacks precipitated by emotional situations which from an etiological point of view, would have been diagnosed as hysteria, but from the character of the convulsion itself, must be diagnosed as epilepsy.

There seems to be little doubt that convulsions of a psychogenic origin with all the characteristic features of hysteria may occur in an individual suffering from idiopathic epilepsy. I have the case of an ex-soldier in mind whose convulsions began following a severe head trauma while on active duty. Besides occasional spells which occurred without any apparent cause he has had three convulsions which were undoubtedly precipitated by severe emotional stress. The first occurred while his company was dedicating a city square to the memory of an ex-service man. The second occurred after he was told that he would not receive an appointment to a certain position which he had been awaiting for several weeks, and the third occurred immediately after he had been notified that the Canadian Government had granted him a compensation which was sufficient to relieve the poverty-stricken condition of himself and immediate family. These convulsions following a stressful emotional state were clinically identical with his other spells and correspond to our present-day conception of the epileptic phenomena.

One may have to deal with a patient suffering not only from epilepsy but a very debilitating neurosis as well. In a study of the industrial history of 47 epileptics who were attending the out-

patient clinic of the U. S. Veterans' Bureau, at Boston, 15 were employed. Of the 32 unemployed the mental attitude toward their affliction, plus the fact that they were being adequately compensated, accounted (as well as industrial conditions) for a large part of their industrial inefficiency. The frequency and severity of spells was far less important than the attitude of the patient toward his disease.

These very commonplace generalities are ofttimes minimized or entirely overlooked in our interest and zeal to find some definite clinical, pathological or biochemical evidence as to what the underlying causative factor of the convulsions may be. Such a scientific approach is justified and commendable. Any medical procedure that falls short of a thorough and complete study when circumstances permit is nothing less than negligent. Yet during the process of investigation much may be accomplished that will work out to the patient's advantage by treating the man while studying his disease.

DISCUSSION.

Dr. Theodora Wheeler (Cambridge, Minn.).—Dr. Thom's paper is comprehensive and its background of social and statistical data is valuable. His broad survey with its consideration of associated phenomena and suggestion of intricate organic mechanism is sound in its ultimate focusing on the individual as the significant unit. This is the only basis on which an intelligent therapy can proceed.

It is often the despair of the institutional physician to provide the individual care which extra institutional workers are able to give their patients.

We have been able to apply a method which provides an improved definition of the individual epileptic's condition.

Two years ago at the Cambridge Colony for Epileptics in Minnesota charts were put into use for 43 patients which show graphically each individual's type of seizure sequence.

The work was done in collaboration with the Mayo Foundation and was undertaken in connection with a study on the ketogenic diet. It may be that others will find these charts of service.

The sheet consists of a graph form which has divisions for the 365 days of the year horizontally and the 24 hours of the day vertically. The entire number and precise time of each patient's seizures for a whole year may thus be entered on one page. This makes possible the comparison and analysis of a mass of epileptic phenomena, the exact detail of which heretofore has been lacking.

Dr. J. Notkin (New York, N. Y.).—I am particularly interested in the emotional aspect of the problem Dr. Thom took up in his paper. Now, there is one group of epileptics in whom the emotional element plays an important

factor. Convulsive seizures are provoked in these cases by emotional stimuli of unpleasant nature, physical exercise, extreme heat and cold, and alcoholic excesses. This group of epilepsies was called by Bratz in 1911 "affectepilepsy," but even in these cases the emotional element is only the precipitating factor which ignites the dynamite. It is true that a psychotherapeutic approach may be of some help for this particular group of patients inasmuch as one can remove the precipitating factor, but I doubt if the same can be said of the large group of the so-called idiopathic epilepsies.

DR. N. W. WINKELMAN (Philadelphia, Pa.).—Dr. Thom has given us a very beautiful example of how to go about handling not only epilepsy but everything in medicine. I would like to ask Dr. Thom about his results. I think that is of interest to all. As far as I heard, Dr. Thom did not give us a résumé of the results that he got in his epileptic cases with this type of technic. I speak of this because in the work that Fay and I have already published on epilepsy, we have found that we can actually control the major epileptic seizures by the control of the fluid intake.

DR. DOUGLAS A. THOM (in closing).—I didn't present this paper to make a record of a specific therapy, but rather as an approach to the problem after ketogenic diets, sedatives, and other methods of treatment in conjunction with other measures that have been applied by the internist have failed. It is simply considering the individual, his problems and his reactions to life in relation to his personality. I don't object to having a patient treated by dehydration, diets, or sedatives while I am approaching the problem in a less specific way.

The only point I am making is so many people feel that we are dealing with an entirely hopeless condition; unless we can find some underlying pathological or biochemical cause, there isn't much to do for it. I haven't any statistics as to actual number of cases. I don't presume I am more successful in treating epilepsy than any other intelligent psychiatrist or internist, but I can cite a great many young people, as I see very largely young people, who were having convulsions very frequently, where there was a home situation, a nerve condition, where there was undue anxiety about work, or an economic situation of some sort, who have been greatly helped by this approach to the problem. I have cited in the body of the paper two or three cases of this type. As I see it, this is an approach that should not only be applied to epilepsy, but it is an approach that should be applied to all disease, to think of the patient as well as the disease, whether it be diabetes, multiple sclerosis or epilepsy, and these other things should go hand in hand with that.

A QUANTITATIVE STUDY OF BEHAVIOR PROBLEMS IN RELATION TO FAMILY CONSTELLATION.*

By JOHN LEVY, A. M., M. D.,
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According to certain psychiatrists size of family and ordinal position in the family are important factors in determining the incidence of behavior deviation in childhood. They believe that the only child is in a dangerous position, developing frequently into a spoiled child. When there are two or more children in the family the interplay of emotions between them, and between the parents and the children, is said to be so intense that prospective parents find it almost impossible to decide whether, after all, it might not be better to have an " only child " rather than have subsequent children face the difficulties of family relationships. So far as the writer knows these assumptions have never been checked up under controlled conditions. This study is an attempt to compare the results of controlled statistical method with what must be called a " hunch " based on clinical experience. While obtaining data to answer these questions other material was obtained which when pertinent will be included in this report.

Unfortunately, the total number of cases studied was only 700. This number, of course, is not adequate for a satisfactory statistical check upon a clinical finding. It should, however, have value as the beginning of an effort to learn whether certain clinical generalizations are supported by statistical evidence. It should also be kept in mind that this study deals only with a clinical population. Similar studies among a non-clinical population (those children whose problems are not serious enough to warrant clinical attention) might produce different conclusions.

The first part of this study was carried on at the Institute for Juvenile Research in Chicago where excellent records lend themselves to statistical investigation. In going over the records the

* This study was made possible through the kind cooperation of Dr. Herman M. Adler, Institute for Juvenile Research, Chicago.

examiner exercised what he considered a necessary selection. All cases having an intelliengce quotient below 80 were excluded so as to avoid the problem of mental deficiency, since behavior problems uncomplicated by mental deficiency were the subject of this study.

Other conditions which had to be satisfied before a case was included for study were as follows: any family which included step-children or adopted children as well as siblings was excluded. Only those children were selected who had been living in their own home at least six months prior to their appearance at the Institute for study, unless they had been sent away from home because of behavior difficulties. Any child now dead was included in the number of siblings if he died after the age of six months. It was thought necessary to establish these conditions in order to be certain that one was dealing with a family constellation in a typical setting. The records used cover a period from 1925 to May, 1928.

Distribution of Problem Children with Reference to Size of Family.

The above table presents the group of problem children referred for study distributed as to size of family. These figures in themselves have little significance. It is only after they are compared with a random sampling of the normal population of Chicago that they can be interpreted. Such controls are exceedingly difficult to obtain. By a stroke of good fortune the writer discovered a research project * under way to discover the size of family of Chicago's population. But even this study did not yield exactly comparable figures. The closest approximation one was able to make to Table I required certain assumptions. Figures are given in the census for "number of members in family, broken and unbroken homes." These figures required a little manipulation before they could be used. Where there were "three members in a family" it was assumed that there would be only one child in that family; four members, two children; and so on. Since broken homes were included in the study of these normal families three

* Access to this study was made possible by the kindness of Dr. Kyrk and Miss Monroe of the University of Chicago. These workers were able to reclassify some of the original data obtained for the latest United States Census.

members in a family might mean two children and one parent, and not two parents and one child. In such cases the three member group would gain at the expense of the four member group. But

TABLE I.

DISTRIBUTION OF PROBLEM CHILDREN IN THE CLINIC GROUP ACCORDING TO SIZE OF FAMILIES FROM WHICH THEY COME.

No. of children in family.	No. of problem children in family.	Percentage of all problem children.
1	91	15.8 ± 1.5
2	146	25.3 ± 1.8
3	116	20.2
4	72	12.5 ± 1.3
5 (or more)	151	26.2
Total	576	100.0

under similar conditions the three member group would presumably lose to the two member group, in those cases where "two members" meant one child and one parent, what it had gained from the four member group.*

It is now possible to compare Tables I and II, Table I representing a clinical population, Table II the general population.†

TABLE II.

DISTRIBUTION OF CHILDREN IN THE GENERAL POPULATION OF CHICAGO ACCORDING TO SIZE OF FAMILIES FROM WHICH THEY COME.

Members in the family.	No. of children in families.	Percentage of all children.
3 (1 child)	5,683	13.8
4 (2 children)	9,060	22.2
5 (3 children)	8,496	20.8
6 (4 children)	6,792	16.6
7 (5 or more children)	10,908	26.6
Totals	40,939	100.0

* It was possible to prove that the number of broken homes has no relation to size of family by comparing the above figures for both broken and unbroken homes against the figures for only broken homes obtained in connection with the economic status of the families—to be discussed later. The relative percentage in each family group whether the home be broken or unbroken was almost identical. Compare Table II with Table IV.

† The figures in Table II do not include those in Table I.

Their agreement is remarkably close. There is only slight variation between the distributions for problem children and normal children in any size family group. The greatest variation is seen in the two and four children families. The percentage of problem children contributed to the clinic group by families of two children is greater by 3.1 per cent than the percentage of children from similar size families in the general population as represented by the control group. Families of four children contribute less problem children by 4.1 per cent to the clinic group than similar size families contribute normal children to the general population. This latter difference is rather high for a chance variation. It awaits confirmation from further similar studies.

Distribution of Problem Children and Controls with Respect to Economic Status of Parents.

An effort was made to learn whether, except for the difference in behavior, the two groups described above were truly comparable groups. It is quite possible that there are many factors which cannot be isolated operating to hide differences. Factors which could be controlled were studied. One such factor is economic

TABLE III.

Economic Status of Parents of Problem Children.

No. of children in family.	Professional.	Executive and official.	Entrepreneur.	Skilled artisans.	Medium and low salaries.	Semi and unskilled workers.	Mean rating.
1	12	8	2	21	16	29	9.0
2	12	17	23	29	27	25	10.4
3	11	14	10	20	16	34	9.6
4 (or more).	11	9	21	27	60	87	8.0
Rating	15	15	15	10	7	5	. .

differences. If "only children" occurred as problems more frequently among the rich, and rich families did not bring their children to the clinic, then this only child group of problem cases would not receive its due quota. The two following tables represent a study of the economic background of the problem group and the control group.

A little explanation is required in connection with the make-up of these tables and the accompanying rating scale. The classification of economic status is the one used in the census. The data obtained for the problem children did not include originally this type of classification. It was necessary to fit the information obtained for problem childrem into this classification. This change has been achieved with some inaccuracy. Frequently one knew the amount of weekly income but did not know the exact economic group. The census' economic grouping is rather broad and uncertain too. The entrepreneur group, for example, might contain people of entirely different social standing. For these reasons it did not seem worth while to make a very close comparison of

TABLE IV.

ECONOMIC STATUS OF PARENTS IN THE GENERAL POPULATION OF CHICAGO.

No. of children in family.	Professional.	Executive and official.	Entrepreneur.	Skilled artisans.	Medium and low salaries.	Semi and unskilled workers.	Mean rating.
1	333	239	657	1162	994	1191	9.1
2	209	184	632	1017	759	1109	9.7
3	93	71	406	692	379	844	9.0
4 (or more).	79	58	543	838	412	1265	8.7
Rating	15	15	15	10	7	5	. .

problem and control groups for each economical group. Instead, a mean rating was obtained for each size family group by allocating arbitrarily a rating from zero to twenty to each economic group. The mean rating was obtained by multiplying the number of children in each economic group by its rating, totalling the ratings for the economic groups within each size family group and then dividing by the total number of children in that family group.

Comparison of these ratings shows a very close approximation; sufficiently close to suggest that wealth does not appear to be acting as a factor of selection in the material under consideration.

NUMBER OF FOREIGN BORN PARENTS IN BOTH PROBLEM AND CONTROL GROUPS.

The question arises whether the larger families may not be of poorer class because of more immigrants among them.

Table V gives the number of foreign born parents among the problem children for each size family group. The increase in the number of such parents as the size of family increases is continuous. One is struck by the exceedingly high percentage of foreigners in the group of largest size families. Since so many of the problem children are referred by social agencies working in foreign sections of the city, one might be skeptical as to how far

TABLE V.

INCIDENCE OF FOREIGN BORN PARENTS AMONG PROBLEM CHILDREN.

Number of children in family.	Number of cases of problem children.	Percentage of foreign-born parents (one or both).
1	90	29 ± 2.7
2	139	39
3	112	43
4	68	51
5 (or more)	143	72
Total	552	48

TABLE VI.

INCIDENCE OF FOREIGN BORN PARENTS IN GENERAL POPULATION OF CHICAGO.

Number of children in family.	Number of cases.	Percentage of foreign-born parents (one or both).
1	4,748	40 ± .6
2	4,002	50
3	2,558	60
4	1,554	67
5 (or more)	1,740	73
Total	14,604	53

these conditions of foreign parenthood apply to the community at large. Fortunately figures of comparison are available. Table VI presents data as to nativity of parent (in this case the homemaker) for the community in general.

One is surprised to learn that in any size family group there are more foreign born parents in the community at large than there are among the problem cases. This fact is especially true in families where there are three and four children. In these two categories the native parents turn a high showing for attendance at

clinic into a relatively higher one. Are the native children in these two groups more likely to be problems? * Or is there some factor which we have not taken into account operating to bring relatively larger percentages of native children from these groups to the clinic? For the present these questions must go unanswered.

Sex of Patients.

Another possible factor working in the direction of selection is the sex of patients. Table VII shows the number of boys and the number of girls in each group of children brought for study. The United States Census Bureau figures show that boys and girls are

TABLE VII.

Sex of Problem Children.

No. of children in family.	No. of cases.	Males.		Females.	
		Number.	Per cent.	Number.	Per cent.
1	91	64	70	27	30
2	146	96	66	50	34
3	116	89	77	27	23
4	72	54	75	18	25
5 (or more)...	151	99	66	52	34
Total	576	402	70	174	30

nearly equally divided in the community at large; there are 104 males to every 100 females in the general population.

According to this study two boys are brought to the clinic for every girl coming under its observation. This difference is hardly disturbed by size of family except in the three and four children families where the number of cases among the girls is smaller.†

Do boys "get caught" more often than girls? Are their problems more overt than those of the girls? Or have the boys more "drive," more freedom of activity, which gives them the opportunity for developing and demonstrating behavior opposed to family and social standards? Whatever else may be the explanation

* The differences here are statistically valid.

† Only a few added cases would be needed, however, to bring these two groups within the two-to-one ratio.

of this point, it would seem well established that size of family is, in general, of slight importance as a determinant of the more frequent visits of boys to the clinic.

Behavior of Children with Reference to Size of Family in a Small Rich Community.*

The question arises whether the relative distribution of problem children according to size of families which was found in a large urban clinical population would also obtain in a small rich community. To answer this question it was possible to make use of a rich suburb of Chicago almost homogeneous in economic and social make-up. The method of obtaining the material for this part of the study was as follows:

Each teacher in a number of schools was asked to submit the names of all pupils in her class. She was then told to draw a ring around those children's names she considered problem children. (The problem should be behavior, and not mental deficiency.) A wide latitude was therefore allowed her. Two criticisms will immediately suggest themselves as to the validity of this method of selection. A teacher might not like to admit she had many problem children in her class. Each teacher has a different idea as to what constitutes a problem child. These criticisms are valid, but they hardly apply to the present study. A trained psychiatric social worker who knows the community very well went through the lists and edited the teachers' work. She too may have had a subjective bias. But there is no reason to assume that any process of selection either on the part of the teacher or the social worker would not act uniformly on all family groups irrespective of size—unless one assumes that certain family constellations produce types of problems more likely to come before the attention of teacher or worker.

Distribution of Problem Children in a Rich Community with Reference to Size of Family.

Table VIII represents the problem population of all the schools studied. Table IX is the non-problem population for the same

* The name of Miss Frances Dummer, Chicago, Illinois, should accompany the writer's in connection with this part of the study.

schools. Both populations are broken up in terms of size of family to which the problem or non-problem child belongs. Comparisons of these two tables brings out the following facts. There is no marked statistical difference between the percentage of problem children and non-problem children in any size family group except in the case of the one child families. In this group the percentage

TABLE VIII.

DISTRIBUTION OF PROBLEM CHILDREN OF CERTAIN SCHOOLS IN HIGH GRADE COMMUNITY ACCORDING TO SIZE OF FAMILY FROM WHICH THEY COME.

No. of children in family.	Number of cases.	Percentage of total.
1	22	10.5
2	61	29.2
3	60	28.7
4	36	17.3
5 (or more)	30	14.3
Total	209	100.0

TABLE IX.

DISTRIBUTION OF NON-PROBLEM POPULATION OF CERTAIN SCHOOLS IN HIGH GRADE COMMUNITY ACCORDING TO SIZE OF FAMILY FROM WHICH THEY COME.

No. of children in family.	Number of children in families.	Percentage of total children.
1	92	5.6
2	460	28.4
3	504	31.0
4	316	19.4
5 (or more)	250	15.6
Total	1622	100.0

of problem children is almost twice the percentage of non-problem children. Unfortunately the small number of cases in this group makes this finding most tentative. It is quite possible, however, that this finding would still obtain, or come out more clearly if the numbers were increased.

In spite of the possibility of the only child being more frequently a problem than children in other size groups in a rich community, the only child when a girl hardly ever becomes a problem. Or

perhaps it might be more correct to say she is not recognized as a problem in such communities. Is this due to the greater inhibiting influence upon girls of such surroundings? Irrespective of size of family, girls in a high grade community are less frequently a problem than girls taken by and large in a mixed grade community. Thirty per cent of the Chicago cases were girls: only 23 per cent

TABLE X.

SEX OF PROBLEM AND NON-PROBLEM SUBJECTS IN HIGH GRADE COMMUNITY.

No. of children in family.	Problem children.		Non-problem children.	
	Boys.	Girls.	Boys.	Girls.
1	21	1	46	46
2	46	15	239	221
3	46	14	272	232
4	26	10	173	143
5 (or more)..	22	8	142	108
Total 161		48 (23%)	872	750

of the suburban cases were girls. (This difference is just equal to three times the probable error of the difference.) Of course it is possible that if we examined only the rich children coming to the Chicago clinic, similar sex differences would be found. The number of such cases examined does not make this approach seem worth while in the present study.

IMPORTANCE OF ORDINAL POSITION AS INFLUENCING NUMBER OF PROBLEM CHILDREN.

The next question that our data enabled us to consider was the importance for behavior deviation of ordinal position in the family. Does the fact that a child is first born, last born, or born in some intermediate position have any influence in determining the incidence of behavior problems? This problem was studied separately in both types of economic and social background. The results for Chicago are given first.

Table XI represents the sampling of Chicago's problem population arranged in terms of order of birth. This table is really a rearrangement of the material presented in Table I. Children from single child families have been excluded however. Table XII

shows how many children there are in the non-problem sample of Chicago's population for each ordinal position.

Comparing these two tables, one finds that first born children are more often a problem, second and fourth born children less often a problem than one is led to expect judging from the numbers of such children living in the community. (The difference found for first born children is statistically valid.) Does the factor of

TABLE XI.

NUMBER OF BEHAVIOR PROBLEM CHILDREN DISTRIBUTED IN ORDER OF BIRTH. (CHICAGO.)

Order of birth.	Number of cases of problem children.	Percentage of total.
First born	179	37.1 ± 2.2
Second born	129	26.7 ± 2.0
Third born	86	17.7
Fourth born	39	7.8 ± 1.2
Fifth born (or later)	52	10.7
Total	485	100.0

TABLE XII.

SAMPLING OF CHICAGO'S NON-PROBLEM POPULATION DISTRIBUTED IN ORDER OF BIRTH.

Order of birth.	Number of cases.	Percentage of total.
First born	10,897	30.9
Second born	10,897	30.9
Third born	6,367	18.1
Fourth born	3,535	10.0
Fifth born (or later)	3,560	10.1
Total	35,256	100.0

"first bornness" lead to the development of problems, or is it as Dr. Curt Rosenau says in a very excellent similar study, a question of age? (The first born child has had a longer time to develop problems.)

Believing the matter of age important, Dr. Rosenau has made a "correction for age." However, he still finds the same difference functioning in an attenuated form. In connection with the need for correction for age, two questions arise. Is such a correction

necessary at all? Is it necessary in the present study? Perhaps the best way of getting at the importance of age for incidence of behavior problems is trying to envisage how it operates. One way in which age can work as an unequally distributed selection factor is through the production of more first children, namely in those families where there is only one child in the family, or where the second child has not yet been born. In this present study all "only children" in both problem and control groups were excluded. (However, having as a control the number of non-problem "only children" in the community, and knowing by this study that "only children" have no greater incidence of behavior disorders than children in large families, it would have been legitimate to have retained both groups.) Another way in which age might act as a

TABLE XIII.

MEDIAN AGE OF PROBLEM CHILDREN, DISTRIBUTED AS TO ORDINAL POSITION IN FAMILY.

Order of birth.	Number of cases of problem children.	Median age for group.
First born	175	11 yrs. 6 mos.
Second born	129	11 yrs. 2 mos.
Third born	82	11 yrs. 7 mos.
Fourth born	38	11½ yrs. 0 mos.
Fifth born (or later)	48	11 yrs. 9 mos.

factor of selection is in the case of very young children. The first born child might reach an age of three or four and could therefore be brought to the clinic for study. His younger brother, two years old and a feeding problem, would be considered too young for study. Does correcting for age meet that problem? That is a problem for which a correction would have to be made for the selecting action of clinics and not for the selecting action of age. However, there is a pre-school division of the clinic in Chicago which did study children three years old or less—some of them were even two years old—and were included in this study whenever they appeared. Moreover, untreated problem children below the age of two are likely to present problems a little later on when a clinic could reach them. It should also be borne in mind that the clinic imposes an upper age limit which excludes first born children too old for study.

Does time enter into the causation of behavior difficulties? Are childhood behavior problems a product of age at all?

From Table XIII it will be seen that the third born child manifests difficulties a little later than the first born child—judging by the median age of the different groups studied—and the second born child presents problems a little earlier than the first born child. Time does not appear to play the rôle ascribed to it. A child of five

TABLE XIV.

NUMBER OF PROBLEM CHILDREN IN A RICH COMMUNITY DISTRIBUTED IN ORDER OF BIRTH.

Order of birth.	Number of cases of problem children.	Percentage of total.
First born	62	33.1
Second born	71	37.9
Third born	35	18.9
Fourth born	10	5.3
Fifth born (or later)	9	4.8
Total	187	100.0

TABLE XV.

NUMBER OF NON-PROBLEM CHILDREN IN A RICH COMMUNITY DISTRIBUTED IN ORDER OF BIRTH.

Order of birth.	Number of cases.	Percentage of total.
First born	522	34.1
Second born	522	34.1
Third born	292	19.1
Fourth born	124	8.2
Fifth born (or later)	70	6.5
Total	1530	100.0

can be a greater problem than a child of ten. One might even argue that the first born child, having lived longer, has had more time to grow out of his difficulties. Another interesting finding in connection with the importance of age is the fact that in this series, the median age for each group of problem children, irrespective of size of family, is about eleven and a half years. One could hardly have been more successful in picking out comparable age groups. In concluding this point of age, the important consideration would

seem to be not the age of the children but the number of first born and second born children in the community from which problem children can be selected, and against which the number of problem cases in each ordinal position can be compared. These control figures have been given in Table XI.

Does the factor of wealth have any influence upon the incidence of ordinal position? Tables XIV and XV give data with which to answer this question.

Study of these tables shows that the differences demonstrated in a more general sampling of behavior problem children have disappeared. No longer is the first born child a problem more frequently than the second born child. In higher class districts the second born child appears to develop behavior difficulties more frequently than the first born.*

Importance of the Sex of the Siblings Nearest to The Problem Child.

Another question worthy of study is this: does the sex of the sibling immediately above or below the problem child have any bearing on the incidence of the problem children of either sex? Expressed concretely: is a boy with an older brother likely to be a problem child more often than a girl with an older brother? This question is a pertinent one in the light of possible fixations between parent and child, and the possibility of this relationship having a radiation effect upon other children. If the first child were a boy who developed a very strong attachment to his mother, this attachment might not be so devastating to the subsequent child, if it were a girl. She would be left free to attach herself to her father!

It has been found that where the problem child is a boy, the nearest sibling above the problem child is a boy in 48 cases and a girl in 26 cases; the nearest sibling below is a boy in 41 cases and a girl in 36 cases. Where the problem child is a girl, the nearest sibling above that child is a boy in 14 cases and a girl 19 cases; the nearest sibling below the problem child is a boy in 24 cases and a girl in 12 cases.

* Unfortunately the number of cases is small, and the differences do not stand up under statistical attack. The point is worthy of further study.

These figures are given for their suggestive value only. In their present incomplete form discussion of them is hardly worth while. They do seem to call attention to the value of a more elaborate approach along similar lines.

Are Certain Behavior Problems Typical of any Special Size Family?

The only child is regarded in the literature as a special type of problem, the problem of the "spoiled child." An effort was made to verify this assumption by classifying the various problems for which children were referred for study, and then finding the distribution of such problems among different sizes of family groups.

TABLE XVI.

Number of Different Types of Behavior Problems Distributed among Families of Different Size.

Size of family	Personality and emotional problems		Habit training problems		Scholastic difficulties		Delinquencies	
	No. of cases	% of all problems	No. of cases	% of all problems	No. of cases	% of all problems	No. of cases	% of all problems
1 child	31	23 ± 3.6	22	17	28	22 ± 3.6	50	38 ± 4.2
2 children	73	36 ± 3.3	39	20	34	16	57	28 ± 3.4
3 children	44	30	22	15	26	17	57	38
4 children	25	30	13	15	13	15	34	40
5 (or more children)	49	25 ± 3.1	27	14	24	12 ± 2.3	98	48 ± 3.5

In the matter of classifying problems, a purely descriptive classification was devised. This method is open to obvious criticisms. It should be possible, however, to catch within the "emotional and personality problems" the "spoiled child" which is itself but a descriptive phrase. Other problems coming within this same group are temper tantrums, moodiness, sensitiveness, sulkiness, difficulty in getting along with other children, etc.

The second group contained the early "habit training problems." Examples of these are feeding, sleeping and elimination difficulties. In this group were also placed bodily disturbances, such as, convulsions, tics and spasms. The third group, "scholastic difficulties," included children who had trouble making their grades, and who suffered from special educational disabilities. Into the fourth group, "delinquencies," when stealing, sex problems, truancy and lying. Children from Chicago were alone used in this special study.

The following facts stand out as important in Table XVI. Two children families produce more emotional and personality problems than any other size family group. One child families produce problems of this type the least frequently.

Is the danger of parental fixation more important when complicated by the presence of a single rival, brother or sister?

"Only children" are troubled much more frequently by scholastic difficulties than children with brothers or sisters. This type of difficulty seems to disappear in direct proportion to the increase in the number of siblings. Since most of the children with scholastic difficulties are referred directly by schools the possibility of the selective action of parents—the parent of an only child might consider inability to spell a more important problem than would the mother of five children—is excluded. Perhaps the rivalry, and occasional help, from other children in the family can act as a desirable stimulus to study and scholastic progress.

Social delinquencies are the "only child's" chief misdemeanor. This type of difficulty occurs much more frequently in this size family group than does undesirable personality deviation. It occurs much more frequently before a sibling comes along. Families of two children live in an atmosphere conducive to emotional stress and strain; families of one child live in an atmosphere conducive to anti-social conduct. Does another member in the family act as a restraining influence against stealing or truancy, the danger of being "told on" at home being so much greater? The largest number of delinquencies take place, of course, among the largest (five children or more) families. But here a different influence is operating; namely the factor of gang activity. The largest families are chiefly foreign, and of poorest economic and social status. They live in crowded sections of Chicago where gangs and delinquency flourish, where an entirely different concept of social life obtains.

Conclusions Drawn From the Present Study.

It is possible to summarize the following conclusions in the light of evidence presented by this paper:

1. In a clinical survey of the population of Chicago the distribution of children's behavior problems appears to be for the most part independent of size of family.

2. In a small rich community, families in which there is only one child may produce problem children more frequently than other size family groups—after due consideration of the number of each size group in the community.

3. The above finding is true only of boys in such communities: only girls do not appear to be often recognized as problems in such rich districts.

4. In a large city boys come to the attention of the psychiatrist more than twice as often as girls. This ratio is somewhat higher in a small rich district.

5. In a large city the first born child is a problem child relatively more frequently than children in any other ordinal position. This finding holds after allowance has been made for the fact that there are more first born children in the community.

6. The second born child is a behavior deviate relatively more frequently than children in other ordinal positions only when a small high grade community is studied.

7. The sex of the sibling nearest in age to the problem child may have a bearing upon the incidence of these problem children.

8. The only child does not appear to be a spoiled child as frequently as children from two children families—if one accepts personality and emotional difficulties as the outward manifestations of a spoiled child.

9. Only children are troubled much more by scholastic difficulties than children with brothers and sisters.

10. The only child commits more acts of delinquency than children with one brother or sister. But most delinquents appear in very large families where economic and social conditions are more important than family inter-relationships.

Suggestions for Further Study of Family Constellation.

As one works through the above material certain suggestions stand out in one's mind as to the value of further work along similar lines. To make studies of this kind really worth while it would be advisable to begin with:

1. Similar studies on a much larger scale in other large urban centers, and in small rich communities.

2. Enough data would be obtained to study the rich children in large centers too.

Other problems growing out of this work which seem worthy of study are:

1. Are family inter-relationships more important than group traditions in determining the incidence of behavior deviation?

2. Is intelligence more important than strictly environmental factors in explaining qualitative differences in behavior?

3. What influence has the sex of nearest siblings upon behavior?

4. What difference is there in the behavior of children of different sex: what are the reasons for these differences?

5. What relationship do children's problems have to parents' problems?

6. What importance has race upon behavior in children? *

* I am greatly indebted to Frederick W. Brown, Director, Department of Information and Statistics, National Committee for Mental Hygiene, for his valuable suggestions and criticisms of the above study. Many of the former have been used.

A STATISTICAL REVIEW OF CONVULSIVE DISORDERS IN THE UNITED STATES.*

By HORATIO M. POLLOCK.

Statistics relating to convulsive disorders in this country are just emerging from the primitive or pioneer stage. Two years ago there was introduced in the state institutions for epileptics in this country a new statistical system. Such system provided for the recording and reporting of data concerning admissions, discharges and deaths in a uniform manner in the several institutions. Some of the institutions have already issued statistical reports in accordance with the requirements of the new system and it is probable that within a few years good statistical data concerning institutional care of epileptics will be available.

In 1922, the Federal Census Bureau collected data concerning admissions, discharges and deaths in institutions for epileptics and on January 1, 1923, made a special census of the patients resident in the institutions. This was the first national census of epileptics and is the principal source of information concerning the problem of convulsive disorders in the United States.

In 1926 and 1927, the Federal Census Bureau collected data from state institutions including those caring for epileptic patients. In 1929 the statistical office of the National Committee for Mental Hygiene collected data concerning movement of population from most of the institutions caring for epileptics in this and foreign countries.

We therefore have a considerable quantity of statistical material dealing with epileptics in institutions.

The available data relative to convulsive disorders in persons outside of institutions are fragmentary and unsatisfactory. The voluminous publication issued by the Surgeon General's office, following the late war under the title "Defects Found in Drafted Men" sets forth the number and rate of epileptic cases found by

* Read at the eighty-sixth annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Washington, D. C., May 5-9, 1930.

boards of examiners among the men drafted for the United States Army in 1917 and 1918.

We also have data assembled by surveys of various kinds and a large variety of estimates.

Using the sources of information as above outlined, we shall attempt to give statistical answers to six simple questions that would naturally be asked by persons interested in the convulsive disorders.

1. *What proportion of the general population is suffering from convulsive disorders?*

In the absence of a general census of convulsive disorders in the population or of representative comprehensive surveys in various parts of the country, a satisfactory answer to this question cannot be given. Dr. A. J. Rosanoff made a thorough survey of mental disorders in Nassau County, New York, in 1917, under the auspices of the National Committee for Mental Hygiene. He found in this county of about 116,000 inhabitants, 1592 mentally abnormal persons. Of these, 72 were classified as epileptic, 394 as insane, 634 as feebleminded and 492 as psychopathic. The rate of prevalence of epilepsy was 62 per 100,000. If this rate were applied to the entire population of the United States at the present time, which is estimated at 125,000,000, it would give 77,500 as the total number of epileptics. This total, however, is very much less than the number that would be obtained by using the results published in "Defects Found in Drafted Men." In the latter study the ratio of men suffering from epilepsy to the total number examined was 5.15 per thousand. In considering this rate it must be remembered that it applies only to a selected group of the population. It seems probable that the rate in such group was much higher than would have been found had the whole population been subjected to a similar examination. The wide variation of results in the various states raises doubt as to the uniformity of standards and methods of examination used by the draft boards in the several states. For example, the rate given for the State of Vermont was 12.7 per thousand while that for South Dakota was 1.2 per thousand. On the face of these returns the rate of epilepsy in Vermont is 10 times as high as the rate in South Dakota.

In summarizing the data with respect to drafted men, the authors of the study state that "epilepsy seems to be commoner in the

older settled states, but this may possibly be due to the fact that the examination was perhaps more critically made in these states." In the light of other data I think we may fairly raise the question as to the accuracy of the diagnoses in many of these older states as the ratio seems higher than would reasonably be expected. If we applied the average results reported by the examinations of drafted men to the general population of the country, the total epileptics in the country at the present time would number 643,750 or more than eight times the number computed from the results of Dr. Rosanoff's survey. It seems probable that the one number is somewhat too low and the other very much too large.

Various surveys and studies in foreign countries give widely different results. Some of them indicate a rate of epileptics of less than 2 per 1000 of general population. The true figure for this country must await future studies.

2. *How many patients with convulsive disorders are being cared for in institutions in this country?*

The Federal census of institutions of January 1, 1923, above mentioned, shows that there were on that date 24,018 epileptics in institutions in the United States. Of these, 12,936 were in institutions for feebleminded and epileptics; 10,016 in hospitals for mental disease and 1066 in almshouses. In the 9 state institutions exclusively for epileptics, on January 1, 1923, there were 7556 patients. At the end of the fiscal year of these same institutions in 1929, the patient population had increased to 9144. Since 1923, a new state institution for epileptics has been established in Pennsylvania. If the average rate of increase in all institutions caring for epileptics is the same as that shown for the 9 state institutions, the total number of epileptics in institutions in the country on January 1, 1930, would be approximately 29,000, or 23.2 per 100,000 of population. Here again, accurate data must await a more complete census. The institutional problem caused by convulsive disorders is only about one-tenth as great as that caused by mental disease and only about one-fourth as great as that caused by mental deficiency.

3. *What is the annual rate of incidence of convulsive disorders?*

We cannot give even an approximate answer to this question. The number of first admissions to institutions for epileptics in 1922 was about one-sixth of the number of resident patients. If the

same ratio obtained in all institutions caring for epileptics the total epileptic first admissions to such institutions in 1929 would have been approximately 4800. This number constitutes only a fraction of the new cases occurring annually in the community—how large a fraction cannot be estimated with any degreee of assurance.

4. *Are convulsive disorders more prevalent in urban than in rural districts?*

In the census of first admissions to institutions for epileptics taken in 1922, it was found that the general rate of first admissions per 100,000 of population in urban districts was 1.7 and in rural districts, 0.9. The rate for males was 2.1 in urban districts and 1.1 in rural districts. The rate for females was 1.2 in urban districts and 0.6 in rural districts. Differences in these rates do not indicate that epilepsy is more prevalent in urban than in rural districts. It would seem probable, however, that convulsive disorders would be aggravated by the stress of life in cities and that adjustment for those afflicted would be more difficult. It is also believed that convulsive disorders from environmental causes are more prevalent in cities than in rural districts.

5. *Are convulsive disorders more prevalent among males than among females?*

Of the 8777 resident epileptic patients for whom schedules were received in the Federal census of January 1, 1923, 4741 or 54 per cent, were male and 4036, or 46 per cent, were female. There was considerable variation in the sex distribution of patients in the several states. Indiana reported 416 males and no females. Illinois reported 180 males and 59 females. Kansas, Massachusetts, Michigan, New Jersey and Pennsylvania reported more females than males.

Of the 1421 epileptic first admissions for whom schedules were obtained in this census, 900, or 63.3 per cent, were male and 521, or 36.7 per cent, were female. Of the 1950 epileptic first admissions to state institutions reported by the Census Bureau for 1927, 1140, or 58.5 per cent, were male and 810, or 41.5 were female. Notwithstanding these rates it is improbable that the rate of incidence of convulsive disorders is higher among males. The excess of males among first admissions is probably due to the fact that the female lives a more sheltered life and consequently finds less difficulty in meeting the demands of family or community life.

6. *Are convulsive disorders increasing?*

From data at present available, we are unable to answer this question positively. It appears probable, however, from the incomplete data at hand that epilepsy is becoming relatively less of a problem in the United States. The census of epileptics taken by the National Committee for Mental Hygiene, January 1, 1920, showed that, exclusive of the epileptics included among the patients with mental disease, there were on the date of the census 14,937 epileptics under treatment in institutions in the United States. The census did not include almshouses but covered both public and private institutions for epileptics and feebleminded. The Federal census on January 1, 1923, enumerated 12,936 epileptics in the same classes of institutions, a decrease of 2001 compared with the previous census. The census of 1904 showed 11,652 epileptics in hospitals for mental disease, as compared with 10,016 shown by the census of 1923. The estimate made in answer to question 2 of a total of 29,000 cases now in institutions, if correct, would indicate expansion of institutional care since 1923, but not an increase in total cases.

The patients with epileptic psychoses in the New York civil state hospitals have declined in recent years. The record of epileptic first admissions and of epileptic patients on the books of the hospital for each year since 1917 is shown in the accompanying table:

PATIENTS WITH EPILEPTIC PSYCHOSES, NEW YORK CIVIL STATE HOSPITALS, 1917-1929.

	Patients on books at end of fiscal year		First admissions during fiscal year		
Year	Number	Per cent of total patients	Number	Per cent of total admissions	Rate per 100,000 population
1917	1,223	3.4	142	2.1	1.4
1918	1,214	3.3	146	2.1	1.4
1919	1,156	3.1	178	2.6	1.7
1920	1,117	2.9	149	2.3	1.4
1921	1,169	2.9	198	2.9	1.9
1922	1,203	2.9	178	2.5	1.7
1923	1,146	2.8	153	2.2	1.4
1924	1,118	2.6	116	1.7	1.1
1925	1,123	2.6	162	2.2	1.5
1926	1,113	2.5	133	1.8	1.2
1927	1,136	2.5	162	2.0	1.4
1928	1,177	2.4	165	1.9	1.4
1929	1,164	2.3	164	1.9	1.3

As the epileptic patients with psychoses probably constitute a nearly constant proportion of the total number of epileptics, it seems probable that a decline in the number of epileptics in the community has also taken place. Such decline in convulsive disorders may be associated with the better care of infants, or the lessened consumption of alcohol, or may be due to unknown factors. In view of the serious nature of the disease, it is highly gratifying to observe its decline even though it still constitutes a serious unsolved health problem.

This brief statistical paper indicates how little we know definitely about the social aspects of the convulsive disorders. It also suggests the desirability of more comprehensive and more complete statistics in this field.

DISCUSSION.

DR. WM. T. SHANAHAN (Sonyea, N. Y.).—Dr. Pollock, as usual, has covered the subjects assigned to him so thoroughly that I really find very little to add to what he has presented.

Toward the end of his paper he referred to the fact that a great deal in regard to epilepsy is not known. That brings up the question, what is epilepsy? Until we know what epilepsy is, that is, know more about it than we do at the present time, it will be impossible to find out how many epileptics there are. Because a person has a convulsion of some sort or, a transient disturbance of consciousness, unless that is repeated, there is no manifestation of epilepsy in that individual.

Dr. Pollock is more optimistic than am I, and I think than some others in the work, in regard to epilepsy becoming less of a problem as the years go on. I think that in proportion to other nervous and mental disorders, epilepsy is just as much of a problem, if not more of a problem today than it was years ago.

So far as obtaining a definite and accurate or reasonably accurate census of even those cases in whom frank convulsions appear, I think that more time must pass before such information will be available. In most, if not all, of the state institutions caring for epileptics, these statistical cards and forms and tables which resemble very much those used for the insane and feebleminded are now in use. The committee that prepared these tried to make them as workable as possible, one of the principal difficulties being in preparing the table which had to do with the different kinds or varieties of epilepsy. That table was but tentative and after a while probably will have to be materially revised.

I wish to congratulate Dr. Pollock on so thoroughly covering his subject.

DR. MORGAN B. HODSKINS (Palmer, Mass.).—Mr. Chairman, I would like to agree with Dr. Shanahan that epilepsy is probably more of a problem

now than it was, say, twenty years ago and if I understood Dr. Pollock, the report from the Surgeon General's office that he quoted was from the statistics of the draft board and not from the examination at the camp.

DR. POLLOCK.—It included both.

DR. HODSKINS.—I had charge of examination of recruits in camp during the war and we examined about 170,000 recruits. I know a good many of the draft boards sent all epileptics to the camps and put the matter up to the examining boards in the camps. Frequently I have gotten the history that this individual told the draft board that he had fits, but they said, "Never mind, it might do him good to get in the army." So we had to reject those. I think there are just as many epileptics in the rural communities as there are in the urban centers. The epileptic in a city is more of a problem than in a rural district. If a child on a farm has a fit, no one pays any special attention to it, but if a child in the city goes out on the street and has one or two, someone complains about it and the Society for Prevention of Cruelty to Children, or some other organization, is apt to get that child into a hospital, if possible.

Some ten years ago we made a survey of the epileptics in Hampden County, Massachusetts. That is the county in which our institution is located. We sent a questionnaire to each physician and asked that physician to report all the epileptics that he knew of or had under his care, giving the sex, initials and age. Those that did not reply to the questionnaire we sent a physician to see. If you will allow Hampden County, Massachusetts, to be a cross section of the population, we found that one out of 240 of population was epileptic, making the total number of epileptics in the State of Massachusetts about 12,000. Dr. Elkind using a different method of getting at the problem, estimated the number of epileptics in Massachusetts and he came within 1000 of our figures.

DR. HORATIO M. POLLOCK (closing).—In the latter part of my paper, I discussed briefly the question of the relative increase of convulsive disorders. The data available, though inadequate, do not indicate such increase. These disorders constitute much less of an institutional problem than is generally thought. In Illinois some years ago, agitation was rife for a new institution for epileptics and the propaganda literature stated that institutions for 11,000 epileptics were needed. The state opened an institution for epileptics at Dixon about 1919 and at the end of the first year had less than 150 under treatment. The institution has been operated over ten years and now has less than 500 epileptic cases. In the older states there is an accumulation of these cases, but the new institutional cases are relatively not very numerous.

The volume dealing with defects found in drafted men covered all defects reported by draft boards and by examiners in the camps, whether the men were rejected or not. The average ratio of slightly over five epileptics per thousand was a little lower than that found in Massachusetts. Possibly that represents a fairly correct ratio, but it seems rather high in view of results shown by various surveys.

A PATHOLOGIC CONTRIBUTION TO THE CONCEPT OF NEUROSOMATIC DETERIORATION IN EPILEPSY, WITH RECORD OF TWO CASES.*

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In a previous purely clinical report, the preliminary outline of which we gave two years ago in Cincinnati, and which will appear in full in this month's issue of *Archives of Neurology and Psychiatry,* we described the progressive neurological changes that develop in epileptics in the course of time. We called these progressive changes "neurosomatic deterioration." Derived from clinical observations, this was a purely clinical concept. It was natural, therefore, that we have looked for an opportunity to study the pathologic changes that might underlie the process of deterioration.

Two such cases have come to autopsy and as both of them were provided with sufficient clinical case histories, the brains were submitted to histologic study. The result of this study proved to be of some interest and we will give you a brief account of the most prominent pathologic changes in these cases as regards the origin, nature, and localization of the lesions that were observed.

Clinically, in both cases we dealt with a typical condition of advanced neurosomatic deterioration associated with epilepsy.

Case I was a boy 12 years old, with negative family history and absolutely normal personal history up to the age of 3½ years. At this time (in 1918) he had a severe attack of epidemic grippe from which he recovered without any immediate complications, but a short time later began to have epileptic attacks and in the course of nine years he gradually developed a typical picture of deterioration with generalized muscular rigidity, flexion of head, trunk and extremities, flexion contracture and clasp-knife attitude of lower extremities, pseudobulbar phenomena, and profound dementia. The frequency of epileptic attacks diminished but when 12½ years of age he developed a severe status epilepticus and died in convulsions.

* Read at the eighty-sixth annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Washington, D. C., May 5-9, 1930.

The pathologic study revealed:

(1) Normal condition of spinal cord.

(2) Acute agonal congestion and acute ischemic alteration of nerve cells in the medulla.

(3) A diffuse chronic degenerative process in the brain, affecting more especially the third layer of the frontal cortex and the basal ganglia, especially the pallidum.

The brain lesions could be divided into three groups:

(1) Old inflammatory, largely organized, vascular lesions, affecting the cortex and basal ganglia. This lesion was evident from the tremendous proliferation of capillaries and blood vessels, especially in the pallidum and peduncular-hypothalamic region, thickening and calcification of vessel walls, collection of veins into groups and knots, forming almost a cavernous tissue, as described by Dr. Stanley Cobb in a case of chronic epidemic encephalitis. There was slight perivascular infiltration in the pial vessels. The meninges were thick and often adherent to the cortex. These vascular lesions, partly organized, are probably the result of the meningo-encephalitis that this child had in 1918.

(2) A chronic, but active, process of an atrophic and degenerative nature, secondary to the primary lesions just described. This chronic process consisted of:

(a) Dropping out and rarefaction of nerve cells, especially prominent in the third layer of the frontal cortex and in the pallidum.

(b) Chronic atrophic nerve cells alteration—shrinking of cells, homogenous protoplasm, mucoid degeneration, fatty degeneration, chronic cytolytic changes, affecting practically the whole cerebrum, but most prominent in the frontal cortex, pallidum, and peduncular-hypothalamic formations.

(c) Secondary gliosis—proliferation of fibroglia and of glial nuclei, filling the areas of cellular devastation in the cortex and in the basal ganglia, marginal gliosis, sub-ependymal gliosis, the frontal cortex and pallidum being particularly affected.

(3) Acute congestion, acute ischemic and cytolytic changes of agonal origin confined almost exclusively to the medulla, explains the death in convulsions.

Case II was a man of 66, an alleged alcoholic, without evidence of syphilis. Clinically, patient was a typical case of what Macdonald Critchley described as arteriosclerotic Parkinsonism. He had high blood pressure, enlarged heart, and albumin in the urine. When 51 years old he began to have epileptic convulsions and later gradually developed progressive neurosomatic changes, generalized rigidity, tremor, pyramidal symptoms, and dementia; in other words, the clinical picture that we accepted as a pattern of neurosomatic deterioration in epilepsy. In the terminal period of the evolution he had no convulsions but developed edema, uremic symptoms, eventually anuria, and died in coma.

The histologic study of the brain showed an advanced cerebral arteriosclerosis with particularly severe vascular lesions in the frontal cortex and in the basal ganglia, the third layer of the cortex and the pallidum being most affected. The lesion was particularly severe in the pallidum and peduncular-hypothalamic formations, corpus luysi, substantia nigra, and red nucleus. It consisted of extensive dropping out and rarefaction of nerve cells; in the pallidum the nerve cells were literally swept out, only small scattered islands of pallidal cells remained, mostly in state of severe degenerative changes. The secondary glial proliferation was of two types: first, a fibrous overgrowth without relative increase of glial nuclei—indicative of an old lesion; second, intense proliferation of glial nuclei—indicative of more recent, and more active glial reaction.

The whole cerebrum showed acute changes which were not localized, involving the cerebral cortex, basal ganglia, and cerebellum: These changes were meningeal perivascular and perineural edema, acute ischemic cell swelling, acute cytolytic and caryolytic changes.

In this case, as in the first, three components can be discriminated in the pathological process:

(1) Chronic arteriosclerotic changes of vessels most prominent in the cortex and in basal ganglia.

(2) Chronic degenerative and atrophic changes in the nervous parenchyma as a direct reaction of the latter to the primary arteriosclerotic pathologic process.

(3) The acute lesions consisting of acute ischemic cell changes which can be attributed to the terminal uremia and death in coma.

SUMMARY.

These cases, clinically, seem quite different; however, both show the same essential features: epilepsy and the symptomatologic pattern of neurosomatic deterioration. In the first case—a child—deterioration developed more rapidly and was more destructive, in nine years leading to the ultimate stage of neurosomatic deterioration; namely, profound dementia and cerebral flexion paraplegia. In the second case—a man 66 years of age, with late epilepsy—the neurologic picture characteristic of neurosomatic deterioration developed more slowly, was not so dramatic, and the symptomatologic pattern was one that could be identified with the arteriosclerotic Parkinsonism.

Pathologically, in both cases was found evidence of a primary vascular cerebral lesion which was, in the first case, an acute lesion—meningo-encephalitis; in the second case, a chronic lesion—cerebral-arteriosclerosis. In both cases the primary pathologic

lesion was the starting point of the same chronic degenerative process affecting principally the frontal cortex (third layer) and basal ganglia (pallidum and peduncular-hypothalamic formation). This last chronic degenerative and atrophic secondary process appears to be the most plausible cause of the clinical symptoms of neurosomatic deterioration. The predominant localization of this process in the third layer of the cortex and in the pallidum appear to agree with the fundamental features of the clinical syndrome which was manifested by disturbances in the psychomotor sphere; namely, dementia and progressive rigidity.

THE MECHANISM OF THE KETOGENIC DIET IN EPILEPSY.

A Preliminary Report of Work in Progress.*

By EDWARD M. BRIDGE, and L. V. IOB,
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The original hypotheses of ketosis and acidosis in explaining the beneficial effects of the ketogenic diet in epilepsy can no longer be held valid. It has been suggested that it acts by changing the fluid relations in the body, but definite proof has not as yet been brought forward. Clinical observations on the feeding of infants have suggested that sudden changes from a high fat to a high carbohydrate diet may be accompanied by marked retention of fluid and rapid gain in weight. Careful metabolic experiments by Benedict and Milner (1907) have shown that, all other conditions being constant, a sudden change from a high carbohydrate to a high fat diet may be associated with a marked loss of water and salts from the body. If improvement occurs in epileptic children after instituting the ketogenic diet a marked loss of weight usually parallels the disappearance of seizures.

To further test the possibility of a fluid loss as representing the active principle of the ketogenic diet three children of practically the same age, weight and type of disease (frequent petit mal seizures daily) were starved for five or six days and the intracellular and extracellular fluid losses calculated from the excretion of nitrogen, sodium, and potassium in the urine (see Gamble, Ross, and Tisdall 1923). (A), previously on a normal diet, lost both weight and extracellular fluid in large amounts, and was rendered free from seizures. (B), previously on a ketogenic diet for nine months with good ketosis but no improvement in attacks, showed similar losses of both weight and extracellular fluid and was also rendered free from seizures. (C), previously on a ketogenic diet

* Read at the eighty-sixth annual meeting of The American Psychiatric Association, Section on Convulsive Disorders, Washington, D. C., May 5-9, 1930.

for nine months with almost complete relief from seizures, lost much less weight and almost no extracellular fluid. Her condition remained the same both during and after starvation. This suggests that improvement with the diet is associated with removal of the surplus sodium or extracellular fluid from the body; when no improvement results these surplus stores have not been removed.

It has been observed frequently that fasting may be more beneficial in epilepsy than the ketogenic diet alone. Fasting is not only a high fat diet (body protein and fat are being utilized) but is also a salt starvation. To distinguish between these two factors a normal adult fasted for two days, and at a later date lived for two days on an ash free diet. The losses of weight, intra-, and extracellular fluid were essentially the same in both instances. Another normal adult had similar losses on a sodium-free diet but not on a chlorine-free diet.

Case.	Sex.	Age.	Wt. Kg.	Daily seizures before.	Treatment.	Intracellular fluid loss.	Extracellular fluid loss.	Wt. loss.	Daily seizures after.
K. H.	♀	13	36	10-15	Fasting 5 days	1672	830	3.2	0
E. W.	♀	13	38	5-8	Fasting 5 days	1601	1193	3.2	0
M. S.	♀	12	36	1-2	Fasting 5 days	1126	126	1.5	1-2
E. B.	♂	28	59	normal	Fasting 2 days	765	650	2.03	...
E. B.	♂	28	58	normal	Ash-free diet 2 days	776	896	1.97	...
R. M.	♂	26	66	normal	Sodium-free diet 3 days	+764	887	1.9	...
N. C.	♂	27	81	normal	Chlorine-free diet 3 days	+64	90	1.3	...

• Indicates an apparent retention.

That the acidosis which accompanies fasting may have a similar action is shown by an experiment of Gamble, Blackfan, and Hamilton (1925) in which a normal child was given calcium chloride to produce a simple acidosis with a result similar to that of the above experiments in respect to loss of sodium from the body. Whether or not a pure ketosis has such an action cannot be said.

It is believed that the action of fasting in stopping epileptic seizures is a triple effect of a high fat diet, a salt starvation, and

an acidosis, all of which tend to remove the surplus extracellular fluid from the body. When the ketogenic diet is effective alone the one mechanism seems sufficient. Since the high fat diet may be able to maintain the good effects of fasting although alone it could not produce them, it is believed that a period of fasting should always precede the institution of the ketogenic diet.

The cases here reported are of one type only: children with petit mal epilepsy. Whether or not the findings can be interpreted as applying to grand mal convulsions and to adult epilepsies awaits further experimentation. A detailed account of the findings is to be published later, after a larger series of cases has been studied.

DISCUSSION.

The discussion was opened by invitation from Dr. Dixon Chairman of the Section, by Professor Puusepp of The University of Tartu (Dorpat) Esthonia, who spoke in French, his remarks were briefly rendered in English by DR. WALTER FREEMAN (Washington, D. C.).—Professor Puusepp draws attention to the importance of alcoholism as a factor in the development of epilepsy in the descendants of rabbits. He has administered alcohol in quantities to rabbits and then bred them and produced in the offspring hydrocephalus, and moreover he finds in the brains of these rabbits small nodules of gliosis with scar formation which he considered identical with those described in the human brain in epilepsy. He calls attention to the fact that possibly it is through the hydration of the brain in alcoholism that we produce these convulsive seizures.

DR. TOM WILLIAMS (Washington, D. C.).—Some years ago I published a paper (*Archives of Neurology and Psychiatry*, Edin., 1913) describing the treatment of juvenile epilepsies of not long standing, which astonished me by the consistency with which the epileptic attacks were suppressed. The basis of the paper was that epilepsy was not as most neurologists had supposed in the past, an organic disease of the brain, but was a metabolic assault. The base of that opinion was that we could produce epileptic convulsions in anyone, even in dogs and cats by giving toxic materials like absinthe. The work of Spratling had shown that the seizures greatly diminished if the metabolic condition of the patient were regulated by a low protein diet. I myself felt that the success of my treatment was partly due to a low protein diet.

Then came along the ketogenic diet the interpretation of which didn't seem satisfactory even when patients improved. This paper seems to clear up the difficulties. We have known since the work of Schmiedelberg in pharmacology that the body fluid is regulated largely by the sodium potassium metabolism. He showed very clearly how one could play upon it by increasing and dimin-

ishing the sodium and potassium. M. Fischer in Cincinnati followed out the same line exactly when he showed how to reduce edema, in his great work "Nephritis and Edema." He described how by injections of concentrated salines he reduced the edema in patients, some of whom were convulsive cases of uremia.

The empirical method I employed was very successful in some cases although without success in others. I was at a loss for explanation, and demanded of laboratory men assistance. The research Association of Neurologists devoted a whole session some years ago to the study of epilepsy. There a paper was presented in which the protein elements seemed entirely without effect in the regulation of the crises of epilepsy. So that perhaps the work of Dr. Bridge will make clear to us now why we have failed with low protein in itself, why we have failed with ketogenic diets, and that the regulation of the epileptic may have something to do with the water content of the brain, to be regulated by the sodium, potassium and calcium in the brain.

In my own diet, I insisted upon a large modicum of salines in the diet, not artificial salines given from the drug store, but salines in fruits and vegetables in which not only were sodium and potassium represented, particularly potassium, but in which calcium was largely present and in which manganese was also an important factor. In some cases the sole diet for 24 hours consisted of fruit. In others a whole meal of nothing but cabbage was prescribed. I gave the name "Saline blood" to the first procedure.

Dr. M. E. Witte (Clarinda, Iowa).—I was interested in the principal paper, which has just been read to us and I, besides, particularly have been interested in the discussion by Dr. Puusepp and the little photographs sent around of the rat. This, since I, for a long time, have made a study of the influence of alcoholism on offspring and this particularly from the eugenics standpoint. My attention was drawn to this particular subject amongst the many cases of epilepsy and convulsive disorders to find so frequently the history of an ancestor or ancestors, either on the paternal or maternal side, having been subject to alcoholism. I found amongst the intimate history we obtain in connection with our people, that more particularly the father, grandfather, or still more remote among the ancestors on both sides, has been markedly alcoholic. My finding was corroborated and increased by the writings of Dieulafoy, a French psychiatrist, who wrote a very readable and suggestive book on psychiatry, which had some vogue years ago, though nothing like as much as it really deserved. Briefly, we found that alcoholism produces a weakness in the germ plasm transmitted to the offspring, which shows itself by instability of the nervous system, more particularly in the production of convulsive disorders. Kraepelin, also, voiced his conviction to the same effect. As the years go by, my conviction has become strengthened, and while I do not underrate the pernicious effect of alcoholism as an originator of degeneracy, I more particularly consider it in the front rank as a developer of disorder, more particularly convulsive disorders, in those who

have inherited an unstable nervous organization. I hold no brief for either the wets or drys in this matter; but I have endeavored to keep my judgment unbiased in my search.

Dr. N. W. WINKELMAN (Philadelphia, Pa.).—Gentlemen, Dr. Bridge really has presented a very illuminating paper. We take the credit in Philadelphia for having originated the dehydration treatment of epilepsy. The work was begun, as we known by Fay; it is not a cure; it is a means of control of the major seizures. The work had numerous substantiations. Bauer, Professor of Pediatrics at Jefferson, has had patients on the ketogenic diet without marked effect, has put them on a restricted water intake and has had complete cessation in nearly all his cases. We have felt, and I think a paper that is coming out next month in the *Journal of Nervous and Mental Diseases*, will practically say what we think, that somebody eventually (we may do it; somebody else will, if we don't) will produce a salt or drug which will eliminate the fluid from the body without water restriction. That I think is the eventual better treatment for epilepsy than we have at the present of limiting the patient to a certain amount.

EPILEPSY AS AN EXAGGERATED FORM OF NORMAL CEREBRAL INHIBITION.*

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EXPERIMENTS:
Normal Reflex Responses to Indifferent Stimuli.
Postponement of "Fatigability" and the Spread of Irrelevant Responses During the Progress of Chemical Decerebration.
Diminution of Peripheral Sensibility—Disorientation.
Activation of Composite Central Sensations—Hallucinations—as Manifested in Behavior.
The Convulsion and the Final Stage of Muscular Flaccidity.
INTERPRETATIONS:
"Fatigability" of Normal Reflex Responses to Indifferent Stimuli a False Conception.
Physiologic and Psychologic Reasons for the Existence of Certain Anatomic Arrangements.
The Underlying—Indirect—Causes of the Epilepsies.
THE CONSCIOUS STATE:
Normal Consciousness a Mosaic of Epileptoid States.
The Underlying—Indirect—Causes of Different States of Attention.
RECOVERY FROM INHIBITION AND FROM INJURY:
The Rapid Recovery from the Anesthesia of Attention or of Epilepsy Contrasted with the Slow Recovery from Direct Injury of Nerves.
The Intermittency of the Anesthesia of Attention or of Epilepsy Contrasted with the Continuous Defect Resulting from Direct Injury.
Abnormal Nerve Impulses as Causes of Certain Epilepsies.
THE SEGMENTAL AND SUPRASEGMENTAL NERVOUS SYSTEMS:
The Chances for the Extension of Nerve Impulses in Different Evolutionary Levels of the Suprasegmental System.
CONCLUSIONS.

EXPERIMENTS.

The manner in which a progressive reduction of the sensory, or conscious, state results in a corresponding release of the activity of certain nerve patterns in the central nervous system, is illustrated

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by the following observations on cats under the increasing influence of picrotoxin.

Normal Reflex Responses to Indifferent Stimuli.—For about 15 minutes after the hypodermic injection of a convulsive dose of the drug (½ to 1 milligram per pound of body weight), the animal appeared to be quite normal. When a sound was made behind the animal by clapping the hands, its ears were momentarily pricked up in the manner of the well-known pinna reflex. When a second or two later the sound was repeated, the response was by the same reflex movement, which was, however, weaker than the first. A third repetition of the same stimulus, after the same interval of time, brought forth a still weaker response, and the fourth, none.

When the same experiment was attempted two or three minutes later, the reflex of the pinna was found to have returned with full vigor in response to the first sound. The second sound elicited a weaker response; the third, a still weaker one; and the succeeding sounds, none at all.

This is the behavior of the normal cat in response to indifferent or irrelevant stimuli. The reflex, in such cases, is said to "fatigue" rapidly until it can no longer be elicited, and to return again after a rest.

If the experiment was varied by the application of a slightly less indifferent stimulus than that of sound, the reflex responses were of correspondingly wider extent. Thus, if the cat was tickled gently with a straw in the neighborhood of the ear, the pinna was momentarily strongly retracted and the animal shook its head vigrously for a moment. Upon the application of the same stimulus a few seconds later, the same response was obtained, following which the cat arose deliberately, walked to another part of the cage and subsided to rest. If the stimulus was repeated again after a few seconds, the same response was obtained and, following the reflex response, the cat arose, turned and faced the observer, blinked its eyes at him a few times, then walked away slowly to the farthest corner of the cage.

Postponement of "Fatigability" and the Spread of Irrelevant Responses During the Progress of Chemical Decerebration.—When, 15 minutes later, the hands were clapped at intervals of a few seconds, it was found that the animal responded by a reflex retraction of the pinna at each sound seven or eight, instead of only

three or four, times, as before. After another few minutes the same stimulus brought forth a much stronger retraction of the pinna, which was accompanied by a tremor of the facial muscles. A little later the animal responded not only by a retraction of the pinna and a tremor of the facial muscles, but by a momentary contraction of the entire skeletal musculature—in other words, the animal was apparently startled at each sound.

Diminution of Peripheral Sensibility—Disorientation.—At this time the cat was largely disoriented. The peripheral sensations which, under normal conditions, guide the animal in its movements amidst its surroundings, appeared to have deserted it. The cat no longer knew the limits of the cage, and it would frequently bump into the wire wall when compelled to move away from where it crouched. The normal cat, when facing the investigator, upon meeting his eyes, blinked at him. The same cat under the influence of the poison, facing the investigator with its eyes open, appeared not to see him. The cat was entirely insusceptible to the same vocal expression of kindness which under normal conditions elicited the well-known gentle approaches of this animal. In this respect it appeared to be completely deaf. Under normal conditions the cat, as it walked across the cage, carefully stepped over small objects in its way, such as the remains of food, taking especial care to avoid anything wet or soppy, and especially excreta. The poisoned cat appeared to be affected with a peripheral anesthesia of touch even in its most sensitive parts—its paws and nose. For it walked indiscriminately into the objects mentioned, and when it stumbled over a larger obstacle, or slipped and fell on a wet surface, and soiled its nose, it made but ineffectual attempts to wipe clean the soiled, sensitive part, and that mainly by movements of chewing and swallowing.

It was clear that although stimulation was highly effective in eliciting seflex responses of a certain kind, the responses were unsuited to the surroundings. The inherent activity of the automaton was no longer guided and modified by that normal accompaniment of stimulation in the higher animals, which is known as peripheral sensibility.

Activation of Composite Central Sensations—Hallucinations—as Manifested in Behavior.—Although there were numerous reasons for the conclusion that the function of elementary periph-

eral sensation was largely absent, there was no reason for a similar conclusion regarding those composite sensory functions which are known as imagery and hallucinations. For while these animals stumbled or fell over slight obstacles, at the same time, in a number of instances, they manifested as amazing degree of co-ordination in the execution of very complex movements integrated and organized into acts. If movement is, at least indirectly, the result of stimulation, the movements of the animals in question were certainly not the result of any stimulation immediately imparted from the outside. For, organized as they were into very complex concrete acts, they were utterly out of correspondence with the present surroundings. The orderly series of activities must, therefore, have been initiated by a correspondingly orderly series of stimulations from within the animal itself. With the addition of certain movements of the muscles of the organs of speech, such a series of orderly activities is the only proof of the existence of the composite sensations of thought, imagery and hallucination in human beings. And there is no reason why a similar series of organized stimuli arriving from within the cat, which are proluctive of a corresponding series of organized movements, may not be called by the same names—thought, imagery or hallucination.

If the foregoing mode of reasoning is true, then the movements exhibited by some of the animals during this stage of poisoning, were in response to hallucinations. The sensory patterns of which such hallucinations were composed, however, could not have been laid down in the lifetime of the animal; for a number of these domesticated cats behaved like their ferocious precursors of the jungle. The movements were remarkably like those of a wild tiger just captured in a trap. They rushed about, bit the wires of the cage savagely, caught at small objects on the floor of the cage and gnawed and tore at them furiously. And so great was the force of the rush up the wire wall of the cage, and so fine the co-ordination of some of these cats, that they would run for some distance along the ceiling, throw themselves from there on to a wall again, to which they clung with tooth and claw. An attempt to pet the cat at this stage of the experiment resulted in a wild escape with all the manifestations of extreme fear, as if the human touch were one of imminent mortal danger. When gently poked with a stick, most of the animals paid no attention; but in one case, when an attempt was

made to pry the cat out of a corner of the cage where it had wedged itself, the animal sank its teeth into the stick like so many iron nails, and had some difficulty in shaking itself free.

This is true of a number of the animals, the picture varying with the size of the dose administered and with the number of administrations. As a rule, a number of small doses have more frequently the effect of producing the picture just described than does a single large dose. The most striking picture of a reversion to the wild state was obtained by the administration of absinthe by mouth one day, followed the next day by a hypodermic dose of picrotoxin.

The Convulsion and the Final Stage of Muscular Flaccidity.—In most instances five or ten minutes after the cat had responded by a general startled movement to the stimulus of sound, it dropped unconscious in a general convulsion. During these five or ten minutes, however, the seizure could be precipitated by a number of weak stimuli, such as by the application of a relatively weak Faradic shock, a sprinkling of water in the face, and even by a clapping of the hands.

The convulsion itself consisted of complex co-ordinated movements, most of which were recognizable as being organized into definite acts, such as those of leaping, of running, of attack or defense. One of the experimental animals, at the moment of becoming unconscious, leaped from a crouching posture into the air and executed a somersault backwards before landing on its feet. Such a movement, it will be admitted, is not one which the animal has acquired during its lifetime. It must, therefore, be a manifestation of the activity of inherited neural patterns. That, in human beings, the movements, during the seizure likewise represent the activity of inherited neural patterns, was shown by means of photographs, in a previous publication.[1]

For two or three minutes after the convulsion, the entire musculature of the animal was flaccid and stimuli were ineffective to elicit movement.

INTERPRETATIONS.

"Fatigability" of Normal Reflex Responses to Indifferent Stimuli a False Conception.—The term "fatigue," as applied in the fully conscious animal to the fading out of the pinna reflex or to that of the contraction of the general musculature in the phenom-

enon of being startled, in response to indifferent stimuli, is misleading. As the animal becomes disoriented by a progressive loss of its peripheral sensibility, the reflex responses to indifferent or irrelevant stimuli persist for increasing lengths of time. It is not, therefore, fatigue which reduces the strength and finally extinguishes the reflex responses in question, but a condition by virtue of which peripheral sensibility in some way intervenes to make irrelevant stimuli ineffective. Anatomical and physiological facts agree in the elucidation of the nature of this condition.

Physiologic and Psychologic Reasons for the Existence of Certain Anatomic Arrangements.—Upon their entry into the neuraxis, all afferent pathways bifurcate. One of the pathways, which is short, consisting of only a few neurons, passes to the motor region of the brain-stem or the spinal cord. The other, which is the sensory branch, consists of a great number of neurons. It proceeds by a long and very roundabout route through the cerebrum, finally ending in the same region as does the short pathway. The question may naturally be asked: What is the possible biological utility of the branching out of a nerve pathway, if the branches converge again to the same point? The laboratory fact that two nerve impulses propagated along the same fiber are able either to accentuate or to extinguish each other,[3] offers a rational explanation of the particular anatomical structure of the nervous system, as well as of the behavior of the animals which possess it.

The application of a stimulus to a nerve receptor, propagates a nerve impulse along each of the two branches of the nerve pathway. The impulse which travels by the short route, as might be expected, arrives at the final destination first and causes a motor response. Such a motor response was in all probability appropriate in that dim period of the past when the progenitor of the present animal had no large cerebrum to stabilize its individuality amidst the changing conditions of its environment. Under the present conditions such a response may or may not subserve a useful purpose in the life of the animal. The question is determined during the passage of the nerve impulse through the long pathways of the cerebrum. Those pathways have been modified by the sensory experiences of the animal during its lifetime. As the nerve impulse travels along them, it is itself modified in accordance with the particular modification of the pathways. A distant analogy of

such a process is to be found in the modification of the movements of a body when it travels over a surface which has been deformed by the previous impact with other bodies. If past experience has been to the effect that the stimulus is of an indifferent nature, the nerve impulse emerges from the cerebrum in a form which interferes with the impulse traversing the short route. The application of a second stimulus of the same kind will, therefore, either not elicit any response or will elicit a different one. It has been seen that in the case of the normal cat, an indifferent stimulus of a certain kind was effective on three successive occasions in eliciting a certain response, but was ineffective subsequently; and that an indifferent stimulus of a different kind was effective in eliciting the same response only once, and that its repetition resulted in a different response. The entire skeletal musculature of a person may become momentarily contracted when he is unexpectedly slapped on the shoulder by a friend. What is really meant by the term " unexpected " is that the nerve impulse which was initiated by the slap on the shoulder has not yet traversed the pathways of the cerebrum, when the impulse which proceeded along the short route has already arrived at its destination. But the person's general musculature will not become contracted if the slap on the shoulder is repeated by the friend immediately afterwards. For the traveler by the long route will have by that time arrived to interfere. Such an extinction of nerve impulses by mutual interference is a process of true inhibition and has nothing to do with the fatigability of the reflex response. For, as was seen, when the long pathways of the cerebrum have ceased to conduct nerve impulses, the reflex response was repeated a great number of times, with unabated vigor.

The Underlying—Indirect—Causes of the Epilepsies.—In the instance of the experiments in question, a chemical poison was the cause which in some way rendered the cerebral pathways nonconductive. And it is known that certain poisons which appear in the human body as a result of disordered metabolism, are potent to accomplish the same end. Alkalosis, as well as acidosis, excessive pressure by a tumor or by the cerebrospinal fluid, and other agencies to be mentioned later, may result in the production of the symptom-complex of epilepsy. Any attempt to consider such agencies as the direct causative factors of the disorder, however, introduces an insurmountable incompatability with a number of facts. These will be considered presently.

THE CONSCIOUS STATE.

Normal Consciousness a Mosaic of Epileptoid States.[2]—General sensibility, or the conscious state, is composed of elements which are known as states of attention. And each moment of attention, no matter how brief, containing as it does a large extent of anesthesia, fixed postures and automatic movements, embodies the complete series of the epileptic seizure. At certain times the picture of the state of attention is that of the purely sensory minor seizure. Those are the times when the person, or the animal, whose peripheral sensibility is largely in abeyance, who is, in popular language "blind and deaf to the world," is absorbed in the re-experience of past sensory experiences in the form of imagery or thought. At other times the picture of the state of attention is that of the mild major seizure. The person is largely unconscious, and thought and imagery may then attain the sharpness and clearness of hallucination; and to the extent that he is unconscious, his movements are correspondingly automatic. The story frequently told of the professor who, deeply bent upon the solution of a mental problem, walked out in the street in his nightshirt, or fell into a well on his way, is not far from the truth. Most persons have seen, if not the same, certainly similar instances. It will be admitted that that part of the professor's behavior which outrages convention to no purpose and certainly against his better judgment, and subjects him to unnecessary danger, is remarkably like the behavior of the epileptic automaton, of the insane, of the baby or of the drunken man. The popular term, "absent-minded," expressive as it is of the lowest state of man's degradation, implies but little disgrace to the person affected. The reason is that a diminished orientation is habitually accepted as the normal wages of a small but intense amount of co-existing sensory activity of a certain kind. Expressed in wider terms of humanity, the proposition stands out in a still clearer light. It is a fact that persons who are habituated to intense mental or muscular concentration—scientists, artists, inventors, and scholars—are poorly oriented. They are notoriously "impractical" people and are, therefore, easily imposed upon. So true is this rule that any deviation is heralded as an exception.

The Underlying—Indirect—Causes of Different States of Attention.—There is little doubt that the underlying basis of different states of attention may be of a chemical nature. Close analysis, as far as it has been carried, reveals the fact that different states of attention correspond to specific chemical changes in the body in a number of instances. The exhaustion of certain solids in the body, makes for the specific states of attention by virtue of which the person or the animal is largely anesthetic to all stimuli except those which have the attributes of general nutritive substances. The lack in the body of the chlorid of sodium, creates a state of attention in which the animal is especially receptive to the stimuli constituting any of the attributes of salt. And the same is true of the want of water; or of the want or excess of certain hormones in the body in directing attention on objects of sex on any attributes of such objects.

Besides such a chemical basis, physical and mechanical states of the body determine specific states of attention. The adjustment of the center of gravitation of the body when a person walks over a moving surface, or on a narrow board across a chasm or when exposed to cold or heat or rain, furnishes familiar examples of corresponding changes in the states of attention.

RECOVERY FROM INHIBITION AND FROM INJURY.

The Rapid Recovery from the Anesthesia of Attention or of Epilepsy Contrasted With the Slow Recovery from Direct Injury or Nerves.—Although in the underlying causes of different states of attention one thus sees a corroboration of the assumption that similar chemical, mechanical and physical disturbances may be the cause of the epileptic phenomena, the following difficulty is encountered. Changes in states of attention may be exceedingly rapid and the moments of attention correspondingly brief. Yet no matter how rapid the succession of different moments of attention, each succeeding one, implying as it does an anesthesia of a different extent and in a different sphere of sensation from the preceding, also implies a correspondingly rapid degree of recovery from the anesthesia of the preceding state. And it is a fact that when a chemical substance is sufficiently potent to interrupt conduction in a sensory nerve to the extent of producing a certain degree of

anesthesia, recovery is relatively slow. The presence of cocaine or of alcohol in the neighborhood of sensory nerves is an example. And what is true of the process of recovery after the direct action of a chemical substance in interrupting conduction in a nerve, is likewise true of a mechanical force, such as pressure, or of a physical agent, such as the lowering of the temperature of the nerve.

That the epileptic seizure, like the moment of attention, may be extremely fleeting, and the recovery from a most profound and general anesthesia extremely rapid, is illustrated by the following case recently observed in the Vanderbilt Clinic. It exemplifies a large class of familiar cases and it is mentioned here only in order to bring the question at hand into a stronger light.

250196. L. M. M., aged 24, a gas-meter reader by profession, has had minor epileptic attacks for the last seven and major attacks for the last two years. The minor attacks, which recur several times daily are so brief that they do not interfere with the pursuit of his occupation, and he would hardly be aware of having been unconscious during the attack if it were not for the following facts. If, during an attack, he happens to hold something in his hand, he drops it. His jaws snap together and the lower lip is sometimes caught and bitten. Recovery is so rapid that if the lower lip does not intervene between the jaws, he can hear the sound of the teeth snapping together.

The Intermittency of the Anesthesia of Attention or of Epilepsy Contrasted with the Continuous Defect Resulting from Direct Injury.—If the picrotoxin in the animal, or a certain disorder of metabolism, or excessive pressure on the central nervous system in the human being, were directly instrumental in causing the anesthesia of the seizure, that effect must be continuous and co-extensive with the cause and it must even outlast it in the general process of the return of function. As a matter of fact, however, in the continuous presence of such causes, the resultant seizures take place with intervals of complete recovery. Thus in the experiments described, at the end of the seizure, the cat regains consciousness. Its pupils again respond to light. It becomes again quite oriented. Sensory and other stimulation once more produce the normal effects. The reflex response of the pinna to an unexpected stimulus of sound takes place as in the beginning of the experiment and fades out in normal fashion. A little later, however, the response is repeated, with the repetition of the stimulus, a large number of times. A tremor soon became evident. The "startling"

reflex in response to slight stimuli asserts itself next. And by and by the animal drops once more, completely unconscious, in convulsions. And this picture is generally repeated three or four times, until the animal either succumbs to the poison or recovers from its effects.

It will be admitted that the direct and continuous action of a drug on a nerve fiber or of pressure continuously exerted upon it, does not result in seizures, with periods of complete recovery.

Abnormal Nerve Impulses as Causes of Certain Epilepsies.—Moreover, actual injury of the nerve fibers as a direct cause of the seizures, does not account for instances of epilepsy which are sometimes cured by the excision of a small diseased part of the cerebral cortex. Nor does it account for cases which are reported to have been cured by the circumcision of a tight prepuce, or by the removal of other sources of abnormal stimulation. Nor does actual injury of the nerve fibers of the brain account for the reported cases of abortion of a seizure by tying a bandage around the arm when numbness or tingling in a finger gives warning of an approaching attack; nor does it account for the prevention of attacks by the irritation caused by a blister on the skin;[4] nor for the precipitation of one by certain movements.[5]

THE SEGMENTAL AND THE SUPRASEGMENTAL NERVOUS SYSTEM.

The Chances for the Extinction of Nerve Impulses in Different Evolutionary Levels of the Suprasegmental Nervous System.—In the anesthesia of the normal state of attention and in the epileptic seizure, with the attendant activities, it is not difficult to see the same principle in operation in different evolutionary levels of the cerebrum itself as has been seen to exist in the case when a disability of cerebral pathways results in the activity of the lower neuraxis. That principle was assumed to consist of an interference of the nerve impulses, with the result of their mutual extinction. The condition for such an interference was seen to be a nerve impulse which, beginning in one nerve fiber, branches out and proceeds at least along two pathways, which finally converge to the same point. And nowhere are diverging and converging pathways so numerous as in the maze of pathways of the cerebral cortex. So

intricate is that maze that it may be justly considered a source of wonder that any nerve impulse entering it is not extinguished by interference. The fact is, of course, that a vast majority of nerve impulses which arrive at the cerebral cortex *are* extinguished. This is not difficult to conclude from the fact that of the numerous stimuli to which the animal is exposed at any moment, only a few are productive of effects. Of the rest the organism is unconscious. The normal chemical physical and mechanical balance of the cortex must indeed be delicate to permit, among the overwhelming chances for their extinction, the passage of a few impulses. When that fine balance is disturbed by the introduction of poisons, by excessive pressure, by a blow on the head, or by the entry of certain abnormal nerve impulses, as those from the irritation of a wound or other sources, no impulses emerge from the maze—they are all extinguished by mutual interference. The result is anesthesia which begins in the receptive areas of the cerebral cortex. The entry of nerve impulses thus being shut off, the neurons which constitute the continuation of the pathway must be successively involved until the entire central nervous system becomes silent. The order of events in the state of attention, of sleep, of drug anesthesia and of the epileptic seizure, corresponds with the progress of extinction of neural activity. It begins in the receptive areas of the cortex, continues along the association pathways, then along the efferent cerebral pathways, finally involving the motor mechanisms of the lower neuraxis. And as each physiologic unit of these structures becomes silent, the phenomena manifested consist in the activity of the inherent function of those pathways whose turn it is next to be silenced. Thus it is that the highly integrated sensations of thought and imagery in the state of attention, and of hallucination in the epileptic seizure, become active when a number of the elementary sensations—touch, vision, hearing, and others—have been extinguished. When the function of the association systems of the cerebrum has next been disabled, the phenomena are those of the activity of the efferent systems of the cerebrum and the motor mechanisms of the lower neuraxis manifested in posture and in co-ordinated and integrated movement. When these are silenced at last, the picture is one of complete and general insensibility and muscular flaccidity.

CONCLUSIONS.

The phenomena of the seizure, whatever the indirect underlying cause may be, are, therefore, directly caused by that process of interference of nerve impulses, with the result of their mutual extinction, which is known as inhibition. Such inhibition is in itself a normal process, to which the phenomena of thought, imagery and dreams owe their existence. Even under normal conditions there is a very wide range in the degree of facility with which cerebral inhibition takes place, thus making for the fact that some persons can exert their powers of attention with greater intensity than can other persons. Any condition whatever that will disturb the chemical, physical or mechanical balance of the nervous system, may result in a facilitation of the normal process of cerebral inhibition, with the appearance of epileptic seizures as a result.

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THE "NEW" CRIMINOLOGY.

A Critical Review.

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On my desk before me lies a book with a significant title: "The Elements of Crime." The book is published and sponsored by the Oxford University Press—a most reputable concern—and the introduction is by Dr. Wm. A. White, than whom there is no better judge of criminal material. Another introduction is by Dean Wigmore. This, coupled with the neat and apparently systematic arrangement of the material, encourages one to think that we have here a first class contribution to a subject that is sadly in need of good contributions. The title of the book is an ambitious one, and the contents quite inclusive suggesting that the author has gone to the very roots of the problem, and who of us, it may be asked, knowing the total sterility of the present-day criminology, would not welcome a genuine contribution to the subject?

In a review of any book, and especially such as the present one, it is of prime importance to inquire first as to the author's qualifications for writing such book. We take it that the only way to study the problem of crime is first and last through the study of the personality of the criminal; hence the minimum that we would expect from a student of criminology is that he should have made intensive studies of individual criminals. In this respect the author's qualifications for performing the set task fall rather short of our expectations. Years of experience as a prosecuting attorney have no doubt given him an opportunity to witness many criminals; but whether the setting is a proper one to study the criminal is open to question. It is doubtful whether the casual observation and examination of criminals, such as is done in courts, is likely to yield information of the type that will help one to understand the "elements of crime." In particular does one doubt whether the psychology of

* The Elements of Crime (Psycho-Social Interpretation). By Boris Brasol, M. A., Former Prosecuting Attorney of the St. Petersburg Supreme Court. Author of "Methods of Criminal Investigation," etc. Oxford University Press, New York and London, 1927.

a prosecuting attorney is of the type that best suits the spirit of the problem. For it is too often assumed that the primary task of a prosecuting attorney is to defend the state against the alleged criminal and he, therefore, looks upon the latter as being always in the wrong, right or wrong. Such an inherent prejudice and negative attitude seems to us to preclude the possibility of a proper understanding of the problem. Nevertheless, we owe both to the movement and the author a fair and impartial analysis of the work. The scarcity of light on the subject obligates us not to neglect a single ray from whatever source. We shall, therefore, examine at length the author's tenets and conclusions, and more particularly the premises upon which these are built; for if the premises are wrong, the conclusions are of necessity also wrong.

The task of the author is a laudable one. Believing in the more modern thought, he is no longer content with "a scholastic interpretation of crime based upon abstract legal formulas, but seeks to explain the nature of the criminal deed in the light of psychology, biology, sociology, anthropology and economics." In this, he feels, "positive criminology (to which presumably he claims to belong) departs from the classical school which dealt with crime quite apart from the realities of life and in a purely formal and legalistic manner." He thus seeks "to find the underlying cause of criminality, instead of merely registering its external manifestations; primarily, its inner mechanisms and especially its motives." For this, he thinks, we are under particular obligation to experimental psychology (how?) and psychopathology. But here he begs to remark that "we must guard against the unfortunate tendency, especially pronounced in Italy and the United States, of exaggerating the part of mental disorders in the perpetration of crimes." In doing so, he believes, "we are more likely to protect the criminal against society than society against the criminal." He also stoutly believes in punishment, "first, to safeguard society against criminality—the foremost duty of the state, and second, as a means of upholding the authority of the social order."

The author having started out so promisingly, we find ourselves quite disappointed with the last mentioned provisos, since they imply that his interests lie mainly with the social side of crime and not with the criminal himself. Nevertheless, we shall proceed with the examination of the book proper.

I.

In the introductory chapter, "Preliminary Data," the author delimits his subject, stating that he will confine himself mainly to the problem of crime, criminals and criminality; more specifically, with an inquiry into the nature of crime itself—"the genesis and the development of the criminal propensity, and the social factors contributing to its structuralization; the inner mechanisms and the growth of the criminal intent; the psychic contents of the delinquent act; the problem of the criminal responsibilities of the insane"; omitting, therefore, the discussion of the scientific investigation of crime, criminal procedure, punishment and prevention of crime. The purpose of the study, he says, is to gain a clear understanding of crime itself—"its nature, genesis, growth and outward manifestations." He says that "social measures dealing with crime cannot be adequate as long as the nature of crime continues to be obscured. To properly understand the subject he calls to his aid psychology, psychiatry, biology, ethics, jurisprudence, economics and scientific disciplines, coming under the heading of sociology." The first difficulty that confronts one, he says, is the absence of any commonly accepted definition of the term crime. After examining the attempted definitions of a number of criminologists, the author summarizes his views as follows:

In every human being (says the author) along with the social instinct there exists an anti-social instinct or egocentric tendency, which in its extreme manifestations assumes the form of criminal anarchy. This anti-social instinct is particularly found in imbeciles, physical and mental idiots and cretins, also known as "sociopaths"; also in people who partly as a result of imitation, partly because of vicious social environment and perverted education, partly because of morbid heredity, even though possessing adequate mental faculties, develop utterly egocentric characteristics, deliberately joining the ranks of the criminal strata of the population. The egocentric tendency then is the generic cause of crime. Moral degeneracy is a permanent symptom of professional criminality.

Crime is a deed, prohibited and punishable under the laws of a particular state, constituting a wilful attempt on the part of the individual against the existing social order and that factor which controls the coordination of the heterogeneous element of which society is the synthesis.

Crime, as a social phenomenon, is a destructive anti-social force, harmfully affecting the individual, the society and the state. The psychological foundation of the habitual crime is the egocentric or anti-social tendency; the propelling factor in the realization of the criminal idea being the anti-social

instinct itself whether of atavistic or acquired origins. At least three mental attitudes are associated in this tendency, to wit:

a. Defiance or neglect of the existing social order.

b. Following the line of least moral resistance.

c. Absence of any remorse.

The act of the individual aiming at the wilful transgression of its dictates, from the standpoint of sociology, is a crime.

Social friction is the prime cause of crime; and social friction is inevitable in a society whose members are unequal. The egocentric or anti-social propensity, or instinct, if not adequately inhibited, causes social friction and ultimately leads to crime.

When one examines the above, it is found to be a lot of tautology dressed up in excessive verbiage. To be expressed simply the author means to say that crime is a bad thing done against society, that the criminal is a bad man and that criminality is a sign of moral degeneracy (whatever it means) and is due to a criminal instinct (whatever that is). What a remarkable discovery!

After delivering himself of the above platitudes the author next attempts to delimit the meaning of the criminal instinct. The awakening and subsequent structuralization of the criminal instinct, are, according to him, generally speaking, not complex.

Example 1: In the family X there are two children, a girl of eight and a boy of six. Their mother is a somewhat careless woman; she is in the habit of forgetting to lock the cupboard in which sweets are kept. On some occasion the girl, taking advantage of mother's absence, pilfers a bonbon; her little brother watches the girl doing it. The sweet booty is divided between the two, and the incident remains unnoticed. Shortly after this, the experiment is successfully repeated. But then, a precedent easily becomes a habit, especially if the act provokes a pleasurable feeling. Finally, mother begins to take heed of the mysterious disappearance of the candies. She locks the cupboards. However, both the girl and the boy have already acquired the habit of stealing, which to be sure so far has been confined to bonbons. When they find the cupboard locked, they both experience an emotion of profound disappointment which they seek to counteract. The girl knows that candies could be bought in the nut shop around the corner. She has no money, but she happens to observe some silver change on father's desk. Why not utilize it in order to buy candy? Father certainly would not have thought of counting the change; so he will not notice the disappearance of a dime. Candies are bought, and once more the savory sensation is restored. Thus, little by little, the criminal impulse assumes the form of habitual tendency. Of course, in order that it might be converted into a criminal disposition of a more serious nature, collateral circumstances would be required.

What a truly revolutionary discovery! This theory, we take it, should promptly and completely solve the problem of crime. If it does not, it is certainly not Mr. Brasol's fault, for he has pointed out hereat the very roots and sources of crime. That this has come to him not through painstaking investigations of many individual criminals but through theoretical reflection or perhaps merely by a flash of intuition, does not in the least lessen the value of the contribution such as it is. But on examining more closely the paradigm and its implied meaning, we are immediately struck by the inanity of the concept offered. Where, we may ask Mr. Brasol, is there a normal boy and girl who during childhood did not do some petty stealing and pilfering? You and I, dear reader, and all the rest of us have done these things over and over again, were caught and punished for it but we continued doing it just the same for several years until we outgrew it, and other interests captivated our imagination. Yet it did not develop in us the "egocentric" habit and somehow we managed to develop into good and decent citizens, leaving the childish filchings behind merely as a stage in one's normal development. A child is a sort of selfish little animal and guided more by instinct than reflection, he looks upon and appropriates to himself anything in the environment that he can make use of. Not habitually, but even instinctively, he is a little criminal. Training and education and all the love that it gets from his family make him eventually give up the little transgressions. He ceases to steal because he does not want to lose the love of those who have become dear to him, because such loss would cause him more suffering than the transgression would give him pleasure. We challenge Mr. Brasol to submit to us analyses of histories of criminals and prove to us that small pilferings, committed several times and gone unpunished, have actually led one to become a habitual criminal. The idea violates every known clinical experience that one has had with criminals. A deed repeated several times does not become a habit unless it is conditioned by some sort of an emotional drive. It is the latter that is responsible for the development of the criminal reaction. But when that is absent, when the pilfering is done merely as an expression of the child's exuberance and excess of energy that must be discharged in some sort of motor activity, it never turns into a criminal activity. Not the deed, nor the habit then, but the emotion behind that is of prime

significance. And here it is we learn that Mr. Brasol's discussion of the subject is wholly inanimate and amorphous. He deals with reactions and not with human beings that display the reactions; not with human emotions but with cold symptomatic manifestations of the same.

Having delivered himself of the ideas as to how criminals against property develop he has the following to say about sexual crimes:

> Likewise, sexual crimes are often the result of acquired habits and imitations and express satisfaction with some artificial or perverted forms of excitations. The experiment having been repeated several times, it is automatically converted into a habit which might lead to the perpetration of most obnoxious crimes.
>
> Notwithstanding the fact that the external manifestations of impulses in sexual crimes differ in every respect from those described in the preceding chapter, the psychic mechanism, by means of which the criminal instinct is brought to material realization, is identical in both cases.

Had Mr. Brasol taken the trouble to study at least one or two sexual offenders, he would probably have realized the gross inaccuracy of his statement. No group of offenders is known whose morality is higher and whose intentions are better than those of the so-called sexual offenders. Be he an exhibitionist or a pædophiliac, he is forever driven by a blind but irresistible impulse to commit the deed; and the remorse and repentance over the deed is often so great as to lead the individual to suicide. Let Mr. Brasol study at least one homosexual, pædophiliac or other sexual offender and he will soon learn that habit plays no rôle in the development of the reaction. For children, taken by and large, do not particularize their sexual activities until puberty; until then all sexual activities are of nearly equal value to them. Every child had its homosexual stage and equally, every child has taken some part in exhibitionistic and peeping activities. Yet only a small percentage of them become homosexually or otherwise perversely conditioned. If it is a question of practiced deeds, then we should have all become thus conditioned. Had Mr. Brasol the least acquaintance with modern psychiatry (his claims to the contrary notwithstanding) he certainly would not have held on so firmly to his idea that habitual reactions lead to crime. The history of sexual offenders universally shows that it requires a particular constellation of emotional situations to condition such a highly specific reaction as exhibited by them. And contrary to Mr. Brasol's belief, which he

repeats over and over again, it can be shown on the basis of unmistakable clinical data and studies, that these sexual offenders are abnormally constituted individuals who deserve our sympathy rather than condemnation and should be given treatment rather than punishment. But sympathy is not a word that may be found in Mr. Brasol's vocabulary. He is a very severe judge; every criminal to him is a moral degenerate; and from the use of this or other terms one senses that Mr. Brasol carries in him all sorts of accumulated antipathic emotions against the criminal. Not a very promising attitude with which to approach a "scientific study of crime"!

In common with other antiquated notions Mr. Brasol submits a division of criminals into two types; the habitual and the occasional. He states that the elements which go to make up the occasional criminal are mostly external. The reviewer submits that this is anything but true as anyone who has had to do with cases of murder will testify. Murders are often committed under the stress of all sorts of emotional entanglements that have their roots in the very depths of one's personality. To say that such a crime is due to external circumstances is to completely fail to understand the meaning of the reaction. In support of this (his) thesis, Mr. Brasol cites the murder by Raskolnikov in Dostoyevsky's "Crime and Punishment." One would imagine that Mr. Brasol being Russian would understand the meaning of Dostoyevsky's novel; but it is obvious that he completely missed its essential point. He believes that Raskolnikov, stricken by neurasthenia and mental worry, and discouraged by poverty and misfortunes, decided to kill the old woman usurer, whose existence he thinks is meaningless and even harmful, in order to extricate his family from the financial plight. He thus assigns gain as the motive for murder. Yet, the reviewer submits, as anyone with the least degree of psychologic insight could see, that the true motive for the murder lay in the inner psychic difficulties of Raskolnikov. The murder was no doubt a psychic necessity to him, such as it was, and the old woman, was not a source of money but a symbol of an obstacle to overcome. An occasional criminal he is, to be sure, but would anybody but Mr. Brasol be so blind as not to see that the murder was an expression of an inner drama as all the subsequent events of the novel show. But a prosecuting attorney's mind is not very apt to under-

stand the meaning of emotional reactions and in a murder which was an outgrowth of many psychic difficulties, he only sees robbery as a motive. Even that clever blood-hound Porphyry Petrovich, the prosecuting attorney in the novel, showed better insight than Mr. Brasol.

In the second chapter Mr. Brasol attempts to study " The Genesis and Nature of the Criminal Propensity." We have already pointed out the superficiality of his theory of habit as a cause for crime. His next excursion is into the causes of juvenile criminality. He believes that this is due to social disintegration and primarily the dissolution of the family. The family, he says, becomes disorganized as a result of either divorce and separation or death of one parent or both. But, we ask, are not divorces merely expressions and results of the disorganization rather than the causes of the same? It may be safely assumed, he says, that children of such parentage are apt to become criminals unless special care is given them. What proof is there for this as a universal rule? We have known children from divorced families who turned out to be perfectly good citizens and equally, children from " good homes " who became criminals. What Mr. Brasol fails to see is that social conditions alone will not explain the individual crime and criminal; and since social disintegration and dissolution of the family are at present beyond our control, would not an individual approach be more justified? Mr. Brasol further submits, that economic instability, periodical industrial crises, unemployment and poor housing conditions tend to increase the volume of crimes in every country. Really! Then Europe should have much more crime than the United States. But the most significant factor to him lies in " the whole cycle of present day ideas, the systematic ridiculing of religion, the fallacious materialistic doctrine that " might is right." The reader will note that Mr. Brasol is a very respectable conservative, of the old Czarist Russia type who would attribute our social ills to the growth of progressivism. When Bolshevism is mentioned or even only hinted at, Mr. Brasol becomes all upset and agitated.

So much then for the " Genesis of Nature of the Criminal Propensity." We know now no more about it, than we did before; nor does Mr. Brasol.

In Chapter III Mr. Brasol attempts to discuss the rôle of the economic factor in the causation of crime. First he submits that there is a close association between poverty and crime. This we believe to be entirely erroneous and based on a wrong appreciation of the data. The poorer classes furnish a higher percentage of criminals because they are numerically larger; but they do not furnish any larger number than their proportionate share. Again, it is well known, and as Mr. Brasol himself would admit, that the contribution of various classes of crime, in spite of great social differences, is practically the same while like classes are likely to make different contributions; which shows that crime is determined by factors other than or besides social stratification. Later on he states that European countries although yet in the grip of economic crises, nevertheless show a much smaller percentage than the United States. This, he thinks, disproves Marxian theory that poverty is the cause of crime. But it is our recollection that Socialism does not maintain that, but attributes crime mainly to the existing capitalistic system and not to poverty as such.

His second statement is that with increase in prosperity, criminals also greatly increase. This would seem to contradict Mr. Brasol's first statement, but let us take it at its face value. Apparently he thinks of United States which is admittedly prosperous, yet shows great increase in criminality. Mr. Brasol makes here the common mistake of interpreting two coincident phenomena in terms of cause and effect. Just because we have prosperity and increase in crime at the same time, does not at all mean that the former is the cause of the latter. The increase of crime in United States must be sought in factors other than prosperity.

His third economic thesis is the instability of the individual budget. Owing to the seasonal unemployment and other economic fluctuations, with the onset of the crisis, crime increases. The sudden derangement of the budget leads to mobility on part of the individual to maintain his accustomed standards and leads to depression—and "criminogenic impulses are set in motion." This it seems to us, would rather support the Marxian doctrine that the economic system, and not poverty as such, is responsible for crime.

Taking thus Mr. Brasol's thesis as a whole it becomes quite clear that the arguments he submits have no scientific validity and

are of the type that an untrained layman might advance. Discussing elsewhere * the popular as against the scientific view of the problem of crime the reviewer has had the opportunity to point out that it is not poverty as such but the variety of emotional entanglements developed in the family that is of prime importance as an etiological factor in crime. The treatment of the criminal therefore, if it is to be intelligent, must be effected on the basis of the particular etiology and not with reference to the crime, which is only a late and surface expression; there must be a resolution of the conflict and a substitution of sympathic emotions for the antipathic emotions which as a rule dominate the criminal. In such a treatment, punishment, therefore, should not be resorted to as it is only a stimulus for further hatred.

In the discussion of the general problem of the causation of crime, Mr. Brasol quite rightly insists on the multiplicity of causes in the genesis of crime. As a literary example he cites the murder of old Karamazov by Smerdyakov in Dostoyevsky's "Brothers Karamazov," and gives robbery as the main reason for the crime, but also cites some additional reasons—bad heredity, epilepsy, lack of training, bad influence of Ivan, etc., all of which overshadow the impulse to greed. The reviewer submits that as in the case of "Crime and Punishment," Mr. Brasol has here too failed to grasp the psychic significance of the murder of the old man by his illegitimate epileptic son, and that greed has not played the least rôle in the commission of the murder. For the crime was committed by Smerdyakov in blind obedience in the implied wish of his half-brother Ivan, to whom he turned over the stolen money.†

At the trial, circumstantial evidence points to the other brother Dimitri as the murderer. Dimitri, tempestuous and neurotic, may be accused of all sorts of transgressions, but he is not the type that could commit murder, not even of the old monster. Even though circumstantial evidence is against him, a psychological study of him should clear him of the murder. Since prosecuting attorneys are not known to be particularly overburdened with psychological

* See the Psychiatric Quarterly, July 1929.

† For a psychologic discussion of murder in Dostoyevsky's novels see Burchell, S. C., "Dostoyevsky and the Sense of Guilt," The Psychoanalytic Review, April, 1930.

insight into criminal behavior, the prosecution secures conviction and Dimitri is duly sentenced to Siberia.

In chapter IV Mr. Brasol discusses the effect of religion upon criminality. He states in effect.

> With the development of new and complex influences, and of inner demoralization, religion, perhaps more than any other element of social integration, has been attacked by different forces of social disintegration. Since religion does contribute towards moral stability, its disintegration has a direct bearing upon the etiology of immoral conduct and more specifically, the genesis of crime.
>
> Religion is more than an attitude towards Deity, it endeavors to clearly define *modus vivendi* for the social group as a whole and tends to harmonize the conflicting interests within that group.
>
> Throughout ages, social evolution has been permeated and essentially dependent upon the element of religion. . . . .

To further illustrate the essentially beneficial influences of religion, he cites a table which purports to contrast the various antisocial criminalistic trends as egoism, vanity, cruelty, vengeance, greed, sexual license, defiance of social order and malice with, respectively, altruism, modesty, mercy, forgiveness, disregard of material wealth, chastity, obedience to law and malice condemned.

This table demonstrates to him the polarity of the two trends of thought—criminality and Christianity—the evil and the just. Since religion " incarnates the rigid principle of duty and self-restraint," it acts, according to Mr. Brasol, as the main counterbalance of the gamut of egoistic impulses and is a socionomic power arresting the growth of criminal propensities.

At this point one is tempted to ask Mr. Brasol whether religion we see today is or ever was the great moral force he claims for it to be. To be sure, it preaches peace on earth and good-will to men, but was there ever a war in the history of the world that has not been sanctioned by religion? Is it not further true that from times immemorial organized religion stood on the side of darkness and oppression? Who was behind the Holy Wars, the Spanish Inquisition, the St. Bartholomew Massacre or the Russian Pogroms? What, only recently, started the flames and fed the fires of the massacre of 20,000 Chinese by Mohammedans? Religious bigotry and intolerance are by-words used in daily intercourse. As for criminals, clinical studies show that the greater number of criminals are deeply religious, which hardly accords with Mr. Brasol's thesis

that religion is a crime preventative. Mr. Brasol, I believe, will grant that few men ever lived who understood the religious emotion as Dostoyevski did. In his novel " The Idiot " he recites the instance of two peasants, friends, who spent the evening together and were not drunk. One of them was attracted by the silver watch the other was wearing. This watch on a yellow bead chain nearly hypnotized him. He was by no means a poor man, not particularly in need of the watch, not a thief, but rather an honest man. But unable to control himself, he takes a knife and approaching the man from the back and praying fervently: " God forgive me for Christ's sake " he cut his friend's throat and took the watch, praying as he murdered.

On the other hand, atheists and agnostics are not known to be particularly criminalistic. Perhaps the very opposite is true and the greater intellects and thinkers are found to be non-believers.

Mr. Brasol is obviously confusing ethics with religion. Clearly, one can be moral and ethical without being religious and, conversely, one can be religious without being either ethical or moral.

A religious survey of educational institutions, especially colleges and universities, has shown that the higher a group of students is advanced in matters of intelligence and education, the greater is the percentage of non-believers among them. Yet would Mr. Brasol want us to believe that the more intelligent and cultured group furnish a larger percentage of criminals? Hardly—the very opposite is really true.

It thus appears to be a pure assumption that religion is a restraining influence in matters of crime. Even if it be true that many religious people are not criminals, it does not necessarily mean that their good behavior is due to their being religious; nor, on the other hand, is there an attempt made here to show then that criminals who are religious are criminals because of their religion. It is merely to emphasize that there is no direct correlation between the two.

But we have not yet learned the real reason for Mr. Brasol's insistence on religion as an anti-criminal factor. He states:

> **Religion teaches the value of non-material things while rationalism teaches only that which is material and pleasurable.**
>
> **Rationalism is narrow and formal, selfish and sullen in its interpretation of human psychology and carries with it dangerous germs of social disintegration.**

The neglect of disintegration of the religious ideal, deprives youth of that element which alone is capable of conveying stability to its moral convictions.

If juvenile morality be guided exclusively by the shifting experience of rational integration, if it be deprived of religious sanction—a condition of mental emptiness must develop, while the anti-social conviction that "everything is permitted," is liable, sooner or later, to sneak into consciousness, producing an attitude of despair. For this reason one must oppose modern thought with its dogma and of utilitarian rationalism.

Mr. Brasol next discusses the relation of family life to crime. He says:

The utter neglect of religion together with other unhealthy social attitudes . . . . drives both parents and children out of their home. . . . . The gulf dividing parents and children is steadily growing wider. . . . . Contemporary men and women alike, seem to have lost the enviable gift of cherishing the infinite delights of quiet family with its genuine virtues, lasting joys and inner comfort. . . . . Principally the family performed the function of the major stabilizing factor, the place of birth and growth of all moral ideas. . . . . Thus the family too is in a state of demoralization and it is because of this that criminal instincts develop unhampered. . . . . Modern science recognizes the immense socionomic importance of the family and the criminogenic significance of its dissolution. . . . . Hitherto domestic education was closely affiliated with religious instruction. . . . . For this reason the church has always sought to uphold the principle of the family. . . . . The appalling growth of delinquency is in a large measure the result of the dismemberment of the family and the vanishing of religion from the field of social phenomena.

The moral influence of the family upon those juvenile portions of the population is losing ground in nearly every country. . . . . Modern pseudo-scientific doctrines, mostly deriving their origin from the principles of economic materialism, entertain the idea that the family is an atavistic and rather reactionary institution.

The following are the objections to the above statement:

1. It is true that many families are religious and that religion aims to foster family ties. That does not necessarily mean that the two are dependent on each other.

2. The disintegration of the family is not cause of crime, but both crime and family disintegration are effects of other social cause at work. These refer in the main to the centralization or capitalization in the present social system with its urbanization, factorization and subsequent disruption of existing social units.

3. Economic materialism does not teach or preach disintegration of the family. It merely records it as a social economic event, a

part of the great changes that modern society is undergoing. The advocates of economic materialism believe that as society changes the meaning of the family will change mainly in the direction that society will take a more direct part in it.

4. That the spread of socialistic doctrines cannot be held responsible for the disintegration of the family is evident from Mr. Brasol's own statistics. The most socialistic country in the world, Russia, does not appear to have more divorces than the most highly capitalistic country, the United States of America.

At the end Mr. Brasol comes to the following conclusion with reference to the effect of economic factors on crime:

> Here then criminology touches upon the real—not the imaginary—causes of criminality in its present-day militant aspect. It is not the mode of production nor poverty, *per se*, nor prosperity, nor the shape of the nose, nor the brachycephalic symptom, nor any other incidental factor, that generates the phenomenon of crime, but those fundamental destructive changes which take place in the composition of society itself, assuming the form of dangerous processes and threatening the very existence of social order.

These "fundamental destructive changes" are apparently, according to Mr. Brasol, due, in the main, to decrease of religion, and growth of rationalism. And this is called "The New Criminology!"

Next to the factors of religion and family, Mr. Brasol has a bone to pick with education.

> Sociology, he says, regards education as the great power in promoting and moulding collective psychology which in turn is requisite to orderly life and progress in the field of social engineering. . . . . The proper function of education is not so much for the individual himself as for the better harmony and in the interests of the society as a whole. The criminalist should consider education not only as a given system of didactics, but also as the sum total of empiric experiences derived by the individual from the various educational agencies of the social order, forcibly and directly reacting upon the mental and moral make-up of every member of the community. . . . .
>
> But education in its perverted form becomes a dangerous weapon of social destruction with a pronounced pathologic and criminogenic effect upon both the individual and the community of which he is a part. . . . .
>
> With the secularization of the school, the "emancipation of the thought" and the dissemination of primary education, juvenile criminality has not decreased: on the contrary, from year to year, it has steadily been swelling.
>
> Statistical information available corroborates the assertion that the ages between 16 and 19 represent the critical period in the life of the average person, coinciding with or closely following school experiences. . . . . Con-

sequently modern education does not produce constructive socionomic generations in harmony with the social order.

Illiteracy is of little significance from the standpoint of criminality. Rural districts are as a rule more illiterate, but the number of crimes there is, comparatively speaking, very low. Large urban centers where primary education is free and obligatory, criminality is rampant. . . . . The illiteracy among negroes in U. S. A. has decreased while the number of colored prison inmates has increased. . . . . All this shows that modern education evinces a well marked criminogenic effect upon the colored population in this country.

The utter neglect of religious, ethical and esthetical elements in modern education, both in the family and in the school, is largely responsible for the almost intolerable vulgarity of the public taste. . . . . Dexterity of the mind is capable of being utilized for vicious purposes and anti-social ends.

However, precisely because of the predominance of materialistic and mechanical ideology in our system of education, and notwithstanding the fact that in the United States, at least, the growth of material prosperity during the last decade has been astonishing—the volume of dissatisfaction and general restlessness, the sum total of moral wretchedness and mental misery, together with the epidemic of atrocious crimes and the alarming spread of professional criminality—are assuming alarming proportions.

The meaning of all this gibberish is that while education, if properly supervised, works for the good of society, modern education is bad and perverted, for there is too much " emancipation of thought." The statement that with the dissemination of primary education, juvenile criminality has increased, is obviously fallacious. Out of a multiplicity of factors that lie behind criminality, Mr. Brasol picks out one that best suits his fancy and makes it responsible for the whole phenomenon of crime. It is an old mistake in logic of which we have found Mr. Brasol guilty on previous occasions, namely: he interprets two coincident phenomena in terms of cause and effect. " Most crimes are committed during school age; therefore the school is responsible for crime." " Negroes are more educated now, and there are now more criminals among them; therefore education is responsible for criminality among negroes." He forgets that the increase in education among negroes is in the main due to their migration to larger industrial centers and cities where much better educational opportunities are provided for them than in the old South. With the urbanization and industrialization there goes a larger criminality; but why blame it on education? But Mr. Brasol is not concerned with providing the reader with an impartial study of social phenomena. A conservative (to say it mildly) of the old Russia, he has a special

hobby and that is, that modernism in any shape or form is bad, very bad. It is, he thinks, responsible for all our crimes and our misery, for the growth of progressivism, materialism and rationalism; it killed religion, killed the family, killed the old good times when a master was a master and a slave was a slave. But for modernism, Russia would still be a Monarchy and not the terrible socialistic state that it is now; the Czar would still be the Czar, and the Russian, his slave, and Mr. Brasol could still have his old job of solving problems in crime by sending the criminal to jail or gallows. The woes and sighs of Mr. Brasol!

What Mr. Brasol had to say about education in general. holds, he thinks, for literature and art too. Says Mr. Brasol:

> The press and literature are effective means for moulding public opinion and bringing about social accord.
>
> A very considerable proportion of the news space of the ordinary daily paper is taken up by accounts of criminalistic and other anti-social affairs.
>
> The view has often been expressed that journalism invariably plays the part of a criminalistic agency, fostering delinquency impulses and inciting moral weaklings to imitate the crimes depicted in the newspapers. However, proof of the actual harm done is difficult to get at. The dime novels probably have a greater deleterious influence.
>
> An individual crime can be seldom traced to the influence of any single news item.
>
> Vanity being one of the basic characteristics of the habitual criminal, publicity is precisely what he is longing for, and in this respect the press often does perform the function of a powerful criminogenic agency. (But there are other people who are vain, vanity is but a symptom.—B. K.)
>
> The educational value of art and literature is constructive, uplifting and profound. . . . . But in our day decadence of literature is a fact which can hardly be disputed. . . . . Dime novels are not necessarily vicious but sexual fiction of the type of Wedekind's "Awakening of the Spring" and others of similar kind, glorify sex. . . . . The moral deterioration prevailing among the younger generation, sometimes termed the "revolt of youth" according to some writers is also caused by the libido. . . . .
>
> Although in individual instances no connection can be detected, the criminogenic character of sexual literature and art is beyond doubt. . . . . Criminological surveys furnish abundant proof that there is a close, almost organic, affinity between criminality and sex perversions.

Here too, Mr. Brasol raves against the modernistic expression in literature and art, for everything that is modern at once arouses in Mr. Brasol most violent antipathic emotions. To him, it is modernism that is responsible for decadence of literature, degrada-

tion of art, glorification of sex and moral deterioration. . . . ." The criminogenic character of sexual literature and art is beyond doubt." But in spite of all his ravings, Mr. Brasol does not adduce a single proof that changing styles in literature and art are in any way responsible for the increase in crime. The old mistake in logic! Crime is on the increase, and literature and art have changed their forms; ergo, increase in crime is due to changing styles in literature and art. And this is called " Positive Scientific Criminology." What a science! What a crime!

There is, according to Mr. Brasol, a relation between the legislative procedure and crime. We shall not quarrel with him on this point for it is true that inadequate or unwise legislation, merely intensifies social friction, which breeds crimes. " It may even lead to revolution." says Mr. Brasol.

> Revolution is detrimental to the best interests of society, tending to increase the volume of social friction. . . . . The egocentric impulses, deeply planted in human nature, are allowed to develop unhampered . . . . and the delinquent propensity, encountering no obstacles on the path of its growth, is easily converted into criminal reality. . . . . With revolution goes more action and chaotic demolition of the legal foundations. . . . . Strictly speaking, it it doubtful whether such elemental activities can be classed as criminal. . . . . Still their criminogenic effect upon the individual mind is obvious.

Here we merely note in passing that when Mr. Brasol speaks of revolution, he becomes tremendously upset.

Although penology does not fall within the scope of Mr. Brasol's books, he has nevertheless something to say about it.

> In the psychology of the professional criminal the social propensity is virtually non-existent. . . . . His attitude towards law and order is negative and the criminogenic disposition is a fixed psychic state. . . . . In the habitual delinquent the potentive criminal animus is a fundamental and firmly established attitude of mind, derived from previous experiences.
>
> What prevents an individual from committing a crime is fear, constructive social and moral influences, lack of suitable circumstances for the commission of the crime, etc.
>
> The application of punishment is largely calculated to create a threatening or restraining impression upon the community as a whole . . . . although of course neither the policeman nor the penitentiaries are capable of bringing up a generation of law abiding and useful citizens.
>
> Penology is a part of criminalistic science but does not properly belong to the discipline dealing with the etiology of crime. . . . . Some criminologists seek to establish a connection between the particular system of punishments and the statistical fluctuations of delinquency. . . . . Some attribute the in-

crease of crime to the laxity of the criminal courts . . . . but the figures are obviously inconclusive. . . . . The fact is that the process of the formation of delinquent instincts is slightly influenced by the penal system. . . . . There does not appear to be any direct relation between the nature of sentences imposed and the number of crimes committed. . . . . There does not seem to be any relation between capital punishment and the number of homicides.

The fact that but a small proportion of criminals are apprehended, only tends to increase crime by making the criminal think that his chances for being caught are small.

Criminal investigation in the United States is singularly unproductive because of lack of proper officers, absence of registration of criminals, antiquated technique of criminal investigation, the absence of institutions for scientific criminal research, whose function it would be to gather and evaluate legal evidence to be brought before the courts.

Where, we may ask, does Mr. Brasol get his authority for the statement that social sentiments are practically absent in the criminal. From his early statements it appears that he postulates that in his definition, namely, that the very fact of criminality *eo ipso* implies lack of social sentiment. If so, then he obviously begs the question. Does he not know that many criminals are in a severe emotional conflict before and after the commission of the crime, and it is not a question of lack of morality, but of two conflicting emotional drives. Nor is there any evidence for the statement that fear and punishment are deterrents of crime; on the contrary, quite universally, punishment creates a spirit of bitterness, hatred and defiance which only lead to further crimes. The technique of criminal investigation in the United States is, according to Mr. Brasol, singularly unproductive. One would hardly believe that, seeing that our prisons are overfilled and taxed to capacity. I think that the most pressing problem in criminality is not more elaborate methods in the apprehension of criminals, but in knowing how to dispose properly of those that are apprehended. Mr. Brasol would have us think that to solve the problem of crime all that we need is to apprehend all the criminals, throw them into jail, and presto, the problem will be solved. It never dawned upon him that the etiologic factors behind crime will still continue to operate, even if all criminals were to be imprisoned.

And now Mr. Brasol proposes the creation of an elaborate and intensively organized Institute of Scientific Criminology. When we came to this topic we brightened up, thinking that at last we were going to have something practical and worth while, for there

is certainly a crying need for the scientific study of crime. Since in the introduction we were promised by the author that it is the prime purpose of his contribution to seek to explain the nature of the criminal deed in the light of psychology, biology, sociology, anthropology, and economics, our hopes began to run high. An Institute for the Scientific Study of Crime—what a fascination! Picture one, similar in scope and character to the Rockefeller Institute for Medical Research. This Criminological Institute attached perhaps to a large prison. Hundreds of criminals studied from every possible angle. A number of psychiatrists making careful and painstaking studies of the individual criminal in the hope of discovering the basic personality difficulties that have originally conditioned the criminal reaction and thus perhaps curing him of criminal tendencies—as yet a well nigh possible task! Another group of physicians engaged in unraveling the home situations. All the material gathered being correlated by a competent staff of physicians, criminologists, psychologists, social workers, etc. To be sure, lawyers and judges are missing in this Institute, but that of course is understood since its essential purpose and function is to cure and not to condemn.

But alas and alack! As we read Mr. Brasol's outline for the "Scientific Study of Crime," we are doomed to a great disappointment. He appears to have a conception of what a scientific study of crime should be entirely different from what we speculated. This is what he says:

> Conditions should be created which would make the enforcement of existing statutes technically possible. This is not likely to be achieved as long as the general standards of criminal investigation continue to remain inefficient. . . . . Criminal investigation can only be facilitated by scientific research carried out in an orderly manner by professional organizations, especially equipped for that purpose. . . . . Its fundamental duty would be to assist the police force in their singularly difficult work of investigating crimes. . . . . Trained experts of such an Institute would actively participate in the examination, not as mere detectives, but as specialists thoroughly conversant in some particular branch of criminal science. . . . . The examination of the setting in which the crime was committed, the careful preservation and analysis of finger impressions, the study of forgeries, cases of poisoning by bacteria, etc. Thus in the course of a criminal inquiry, many departments of human knowledge should be summoned and called in council. . . . . A very complete "Tentative Scheme of Organization" of such an Institute should provide for 8 large departments, viz: Chemical, Optical, X-Ray, Medical

(morgue, etc.), Mechanical (tools, etc.), Criminology museum (collection of burglars' equipment, forged bills, explosives), etc.

So this Scientific Institute for Criminology simmers down to a highly efficient detective agency that utilizes the contributions of biology, sociology, anthropology, etc., to round up criminals. Bigger and better policemen! Larger and better detectives! But not a hint about studying and understanding the criminal. Verily, it is playing Hamlet without Hamlet in it.

Now our original suspicions are verified. True to the psychology of a prosecuting attorney, Mr. Brasol has not the slightest interest or understanding of the individual criminal. We can just picture him as the Director of the Institute he proposes, directing its activities, hunting down criminals in the most efficient manner. What a project! What a future! Perhaps in the innermost layers of Mr. Brasol's mind, the whole purpose of writing this book was to create for himself a job as a head of a "Scientific Institute for Criminology" such as it may be; for émigré Russians are out of work now and a Director's job is nothing to sneeze at; even counts and princes have gone to work.

And let us suppose that the impossible has happened. Suppose that through the high efficiency of such an Institute, as Mr. Brasol proposes, every criminal has been apprehended, given a severe sentence and then securely locked up behind the bars. And then—and then, the next generation will have its full share and quota of criminals as any other generation ever had. For if we have learned anything at all, we have learned this, that the solution of crime is an extra-legal problem, because its genesis is independent of, and precedes, any laws.

II.

Having thus proven, to his own satisfaction at least, that crime is a habitual reaction due to the overdevelopment of the "criminal propensity" and that of the social factors, the most prominent rôle is played by disintegration of the family, present economic conditions, poverty no less than prosperity, and above all by modernism with all its attendant evils, Mr. Brasol now states that he is going to discuss the delinquent himself because "he is not only the author of the prohibited and punishable deed, but is, as it were, the germ carrier of the anti-social energy, the discharge of which constitutes crime itself."

The author dismisses the claim of the anthropological school of a distinct criminal type, as not having a valid scientific basis. He believes, however, that the criminal propensity can, in a certain measure, be conveyed from generation to generation, although the heredity alone is quite insufficient to explain the origin of habitual criminality.

In the matter of hereditability of physical and psychic features he believes that these can be

> genetically transmitted; but acquired characteristics are not inheritable. . . . . Now it has been pointed out that delinquency in the parents is most likely, in one way or another, to manifest itself in their children; and vice versa, that among habitual criminals, there are many whose parents were either criminals or persons of mental debility.
>
> Statistics show that the heredity factor in crime is relatively small and the figures tend to bear out the general contention that unfavorable heredity is infrequently encountered among criminal strata of population. . . . . It is the combination of heredity and environment that to a large extent explains the phenomena of behavior. . . . . Mental aptitudes are probably not inherited. . . . . Criminality cannot be inborn since crime is primarily a sociological conception.
>
> The delinquent impulse which, as we have been maintaining, is closely affiliated with the egocentric emotions of man, is not a congenital state, but an acquired disposition which prompts the criminal to deliberately ignore persistently undermine or shrink from the social order to which he belongs. The criminal propensity, then, is a habit; the mechanism of its formation is similar to that of the formation of any other habit. It is this problem, therefore, which should occupy the focal attention of the criminologists—not the arbitrary assumption of inheritability of something uninheritable.

Lombroso's theory of inherited criminality has long been shown to be without scientific validity. In the face of overwhelming data to the contrary it would have required on the part of anyone great effort and ingenuity to maintain such a thesis. Nevertheless, credit should be given Mr. Brasol for maintaining the theory that criminality is conditioned on environment; this chapter appears to be the only bright spot in his otherwise dark study of crime. But at the very moment when we were ready to give Mr. Brasol due praise and credit for an enlightened view, we discover a snag. For, further in the discussion we find that the main reason for Mr. Brasol maintaining the thesis on the acquired nature of crime, is to find a basis for putting the blame of the crime on the criminal, because, according to his logic, if crime is a conditioned reaction, it must be a

wilful reaction and therefore must be punished; whereas if criminality were to be proven as being hereditary, then, of course, the criminal should not be punished. But Mr. Brasol must have justice and his pound of flesh, otherwise the world shall come to an end.

The next problem to be examined is "the psycho-physical nature of human conduct in order to form a clearer conception of the complex mechanism requisite to the crystallization of habits in general and morbid dispositions in particular."

> Habit formation depends not only upon instinctive, but on rational elements as well. But habits themselves are crystallized effects of the compound influence of four principal elements: (a) Fundamental Desires, (b) Instincts, (c) Temperament, (d) Character.
>
> The sociologic meaning of crime makes it absolutely necessary to examine the different theories of instincts in order to determine the place of innate tendencies in the etiology of human behavior, mainly, in its anti-social or criminal aspect. At the same time, because instinctive acts are often mistaken for, and confused with, reflexes, it may not be out of place to explain the difference between these two groups of biological phenomena.
>
> While reflexes do not enter into the etiology of human conduct—in the sociological meaning of the term—instincts, on the other hand, occupy a position of singular significance in the formation of individual habits, and, therefore, are of momentous importance to both sociology and criminology.
>
> There are two fundamental categories of instincts: (a) self-preservative and (b) reproductive. . . . . These responses are unlearned and therefore must be innate. . . . . Cognition is absent from them in the early stages of development and may be spoken of as sub-instincts.
>
> The sexual sub-instinct, under unfavorable circumstances, acquires a perverted orientation which in the adult may reach the degree of a distinct psychophysical anomaly (psychopathia sexualis) or become a criminal habit.
>
> There is also a third subsidiary group of instincts, namely, the imitative tendencies . . . . it belongs to the inborn propensities. . . . . The phenomenon of habitual criminals, at least in its rudimentary stage, is largely caused by the factor of imitation. . . . . The child is not a born criminal, but is very selfish and if it constantly observes in his immediate environment, a long series of morbid habits, recited by adults, the egocentric current, inflamed by imitative acts and finding no check in opposite emotions assumes a strong anti-social direction.

The sum and substance of all this is that, according to Mr. Brasol, if an instinct becomes perverted it may lead to sexual abnormality or criminality. We are obliged to accept it on faith, for the author gives us no proofs in support thereof. Similarly, his statement that criminality is due to imitation, is not supported by any data. If criminality is caused by imitation of elders, how will Mr. Brasol

explain the development of a criminally inclined child in a non-criminal family.

Continues Mr. Brasol:

Not all habits which go to make up individual conduct, are derived from or directly dependent upon inherent predispositions. On the contrary, we are cognizant of many habits which are rather remotely, if at all, connected with instinctive responses, being artificially acquired modes of conduct and bearing no influence whatever upon the biological principles of human existence. Some of these customs form no part of the psychic process originating the phenomenon of crime. Such are, for instance, intentionally adopted manners or practices of reading before retiring, wearing certain things, visiting certain people, etc. There is no instinctive foundation in any of these acts, and, furthermore, they stand in no relation to or in conflict with the social order. In the sociologic sense, they are indifferent or neutral facts.

Criminology ought to be guarded against confusing the two categories of habits—the purely instinctive and rationally planned—because when a crime is the result of a deliberately evolved anti-social habit, the jurists stands face to face with a crystallized criminal intent which is often requisite to the consummation of the particular crime itself.

There are numerous artificial habits which do become essential elements in the formation of the criminal propensity. . . . . An act repeated on several successive occasions may grow into a habit. It is not unusual for a person whose previous record was clean, when confronted with some hardship, to commit willingly a dishonest deed which may temporarily solve the difficulty. Now, if the individual succeeds and his transgressions remain unnoticed, he might, on some future occasions, be tempted under the stress of analogous circumstances, to repeat his experiment. If then, the deed should become converted into a habit, it would be scarcely right to call it instinctive.

In some cases, the instinctive element plays no part whatever in the crystallization of the criminal propensity. On the other hand, there are habits which have the element of compulsion as their underlying cause, and, consequently, are devoid of the symptoms of volition. Different acts of this nature frequently come with neurosis.

Typical signs of compulsion neurosis are represented in obsessional functions, like kleptomania and pyromania. These uncontrollable acts are apt to become habitual. Still, from the standpoint of legal imputation as well as in the light of the sociologic notion of delinquency, they cannot be considered crimes owing to the absence of volition in the process of their perpetration. In a way, they may be compared with acts committed under the influence of vise major (force majeure; only in vise major the constraint is originated from without (elemental forces of nature, political upheavals, exercise of somebody else's malicious will, etc.); whereas, in obsessional behavior the factor of compulsion works from within.

A more complex aspect of the same type of involuntary habits is found in a mental disease of a rather obscure nature, known as latent epilepsy.

Psychologists belonging to the Freudian school have taken the view that many instincts exercise a steady influence upon the formation of habits and mental attitude throughout life. . . . . The suppression of natural instincts, is believed by some to affect abnormally social conduct. . . . . It often occurs especially among women, that an instinct is suppressed, not by a restraining factor from the outside, but by an intricate psychic state originated from within. Such feelings, as fear of pregnancy and the specific pains accompanying it, as well as the dread of losing physical attraction, may account for the neuropathological state known as "frigidity." (Parenthetically it may be noted that fear of pregnancy is not a cause of frigidity, but rather a consequence of it. Frigid women, however, may rationalize their frigidity in terms of fear of pregnancy, fear of pain, etc. I would advise Mr. Brasol to read Stekel's work on the subject and inform himself more adequately on it.—B. K.). Unfortunately, the aversion to normal sexual intercourse affects both the female and the male of the family union and sometimes leads to its dissolution.

The suppression from without or the auto-suppression of normal instincts, first, leads to perversions of different kinds; second, that these corrupt habits are easily converted into criminogenic propensities; and third, that such habits, in their extreme manifestations, assume the form of insane practice and obnoxious crimes.

Mr. Brasol's division of habits into instinctive and artificial is not supported by any studies on habit reactions among humans, but is an abstraction entirely evolved theoretically. One may, if he will, question whether habits are at all based on instincts, but since Mr. Brasol admits that many habits are instinctive, his claim that some habits are not based on instinct becomes wholly artificial. He seems to think that if a reaction appears far removed from the instinctive level it is therefore not instinctive. We know, however, that many of our higher activities can be traced to more instinctive patterns. Mr. Brasol's insistence on this artificial division is due to the fact that his pet crime theory is that crime is a wilful act and therefore punishable; and he therefore must have habits that are not instinctively conditioned, otherwise his criminals could not be held responsible. Here we have an exquisite demonstration of how Mr. Brasol juggles facts and develops artificial theories to fit his preconceived fancies. And this is supposed to be a work based on biology, psychology, economics, etc. What a travesty on these disciplines!

Next, Mr. Brasol has something to say on the physical factors in crime.

The influence of bodily defects upon a temperament is a momentous factor in the structuralization of criminal disposition. . . . . Some characteristics

depend upon organ inferiority. In a case of sexual murder, the history of the delinquent having been carefully examined, it was found that, as a youth, while studying in a military school, his class-mates made fun of the underdevelopment of his otherwise normal sexual organs. These derisions became a source of great worry to the boy and made him gloomy and nonsocial. The idea developed in his mind that he was incapable of normal intercourse; on several occasions, this led him to seek gratification of the natural instinct in homosexual practices. Whether under these influences, or because of some undetected facts in his past history, he soon began to exhibit a strong sadistical inclination. Perhaps, this perversion was caused by the antipathic sexual attitude towards women in general, whom he, strangely, blamed for his alleged debility. When having attained the age of 29, he murdered a prostitute whose body was found with some thirty wounds and dreadful mutilations, the location and nature of which, in themselves, were sufficient to prove the sadistic character of the crime. Here, again, we have an instance demonstrating the important fact that physical symptoms, not pathogenic *in se*, may create a temperament which accelerates the growth of anti-social and criminal proclivities.

Thus, the physical element undeniably is a constituent part of temperament and that particular frame of mind which is its habitual complement.

The idea that a physical inferiority may cause one to become a criminal, has no substantial basis. As a matter of fact, crippled individuals, as a group, probably furnish less than their share. Neurosis is much more likely to develop here than criminality. Mr. Brasol, grossly untrained in even elementary logic, argues from part to the whole. The fact that one individual with undeveloped sexual organs committed a crime does not at all mean that the crime was due to that; for similar crimes have been committed by hundreds of people who physically were well developed. Nor, so far as we know, is sadism conditioned on physical anomalies. Stekel, who has written a large work on this subject,* does not seem to share this view, and in going over his material, I cannot find any significant evidence to support the contention. In actual clinical studies on criminals, one finds that physical factors do not appear to play any significant rôle and physical abnormalities among criminals are surely no greater than among the general population. Goring came to the same conclusion; and so have many others.

* Stekel, Wilhelm. Sadism and Masochism. Horace Liveright, New York, 1929.

† Goring. The English Convict.

The last two chapters deal respectively with the problem of *crime* and *responsibility* and *crime* and *mental disease*. As there is considerable overlapping they may be discussed together. As one reads the author's description of mental diseases it becomes perfectly clear that his knowledge of the subject is of second-hand nature and that he probably never saw the inside of a mental hospital, let alone having studied any individual cases. But let Mr. Brasol speak for himself.

Criminology deals with the person of the delinquent, his changing psychic states and temperamental disposition.

In this measure may neurology and psychopathology be able to give us precise information regarding the nature and major symptoms of mental deficiencies. (Mr. Brasol makes psychosis synonymous with mental deficiency—a gross mistake—B. K.)

As psychopathic phenomema are preeminently anti-social, they have, or may have, a common root with criminality (why not follow up this very good idea?—B. K.) and therefore should not be ignored by the criminologist, while the psychiatrist must necessarily take into account the important facts that mental disturbances frequently find their outward expression in prohibited and punishable deeds. (Haven't they though? The psychiatrists were the first to point the relation between crime and insanity—B. K.) Besides, the alienist has a direct and practical interest in the etiology of crime in all cases which involve the plea and raise the legal problem of imputation. (The psychiatrist is interested also in all criminal cases because crime is a psychiatric event and can only be understood through careful individual personality studies.)

The individual crime is, according to the author's understanding, a wilful transgression. . . . . The phenomenon of will is postulated. . . . . The will to commit an act involves the presence of consciousness, the planning and choosing of the act, this leading to the commission of the act.

Having thus expressed himself on the relation between psychiatry and criminology, the author proceeds now to give us his view of psychiatric material proper. His rendition is certainly more confusing than enlightening.

In mental disorder . . . . there is disintegration of the will mechanism. There is difficulty in concentration, attention is disturbed by the least influence . . . . the trains of thought assume an irregular and purposeless motion, strongly interfering with the symptomatic operation of the mind. . . . . In addition . . . . the brain is often invaded by internally arising impressions. . . . .

Many forms of psychopathic sexuality have their origin in this mental condition the reason being that much of the subconscious material is composed of vague sexual urges, ordinarily under control . . . . but otherwise infecting the fields of our consciousness. . . . .

According to Krafft-Ebing . . . . acts of exhibitionists are likely to be committed as the result of neurasthenia as the derangement of the volitional apparatus (irresistible impulse). The consequent conversion of the delinquent tendency into the offense itself, is obviously due to the all embracing decomposition of the volitional mechanism on the one hand, and the psychic condition of dissociated consciousness on the other.

This meaningless jumble of words will hardly give any one a clear picture of what mental disease is.

The discussion of the problem winds up with a reiteration of the author's belief that only those cases of criminality could be relieved of responsibility that show disturbance throughout the entire field of mental operations while if other fields are not so fully affected, the individual should be punished accordingly.

With such ignorance of the significance of psychic factors in human behavior, it would be transgressing the limits of optimism and good judgment to expect anything new and enlightening on the next topic—crime and responsibility.

In the matter of *crime* and *responsibility* the author holds strongly and firmly to the view that barring certain exceptions, every criminal is mentally normal, every criminal act is a wilful and malicious act and that punishment is the main safeguard for society as well as most conducive to the best interests of the criminal. In particular, does Mr. Brasol insist that insanity and criminal irresponsibility are not identical terms. He says:

The development of the anti-social propensity goes together with the atrophy of the social instinct and signifies moral degeneracy. That state of mind, however, must not be mistaken for any form of mental disorder. Occasionally, it is true, we may find the incorrigible offender suffers from some kind of mental disease; but it is an error to maintain that such anomalies constitute the root of criminal phenomenon.

In this respect Mr. Brasol is more in accord with the conservative elements who would punish a criminal even though he is insane. The more progressive and more scientific conception is to regard every crime as expression of abnormal mentality or deranged emotional life. Mr. Brasol is, of course, entitled to his opinion, but let him not parade as a progressive; for although dressed in modern clothes, he is the same conservative that he probably was in Russia. Says, further, Mr. Brasol:

The commission of a crime, *ipso facto,* does not raise the question of insanity of the one who committed it. Daily experiences teach us that the overwhelm-

ing mass of offenses are perpetrated by perfectly normal individuals who fully comprehend the nature of their transgressions and anticipate and aim at their consequences. . . . . The supposition itself that criminals are, or necessarily must be "insane" cannot be held by science and should be emphatically rejected, for many insane never commit crimes and many criminals are not insane. Only in specific cases may a causal condition be disclosed.

In the different psychological manifestations of criminality, the general mental condition of the individual may retain all the characteristics requisite to normal reasoning. . . . . What has to be determined here is the measure of punishment to be inflicted upon the convicted defendant. . . . .

Criminology should be guarded against the philanthrophic tendencies of treating crime merely as a form of psychic disease, and seeking, with the aid of psychoanalysis, to explain various brutal manifestations of recidivism by "suppressed complexes" or "sexual introversion." . . . . There is no truth in the theory that every homicide is necessarily an act of compulsion, or every theft a manifestation of kleptomania. . . . . Inu many cases this policy defeats the fundamental aims of criminal justice and leads to the impunity of the delinquent.

. . . . With reference to responsibility in cases of insanity, it may be said that:

1. An individual may be insane in most phases of his mental life and nevertheless be fully responsible for an act committed either in that province which is not affected by his psychic derangement or committed in the lucid interval.

2. A person may be sane and still be abnormal in some respect which will excuse or mitigate his transgression.

The French code, as with reference to epilepsy, disregards penal responsibility not only at the time of, but as well as between the attacks. According to the author however, epilepsy does not invariably constitute a factor *eo ipso* precluding responsibility; for an epileptic may act with a full measure of reasoning power. . . . . Of course, in various forms of epilepsy, even though consciousness may retain a high degree of normalcy, the volitional mechanism may be disintegrated.

If the law is not closely defined, the jury because of psychiatric ambiguity will, probably, in nine cases out of ten, acquit the defender. (Wouldn't that be awful?—B. K.)

We could understand these statements coming from a layman or a conservative judge, but it is indeed a disappointment that it should come from a man who claims to hold an enlightened view, and tries to give the reader the impression that he is in accord with the recent advances in psychology. The "enlightened view" turns out to be a garb behind which are hidden the most backward opinions on the problem. Here too, as elsewhere, the author merely gives his views in a dogmatic manner, that apparently does not tolerate con-

tradiction, without supporting the said opinions with anything like a reasonable argument.

Mr. Brasol would regard a person irresponsible only when he is in a condition that is obviously abnormal. Should however, the individual commit a crime during a remission, Mr. Brasol would disregard the originally abnormal mental state and punish the man because during the remission he appears normal. Mr. Brasol, who appends a large bibliography on psychiatry, has apparently failed to profit by the reading. He still holds to the view of "partial responsibility," that "a person affected by some fundamental psychic defect, may still be capable of distinguishing between 'right and wrong' and therefore held responsible." Has not Mr. Brasol learned that this partial obvious psychic defect is only the upper part of the mental picture and that buried beneath are all sorts of psychic abnormalities that are not so obvious? Has he not learned, what psychiatry has learned long ago, that an individual may know intellectually between right and wrong, yet unable emotionally to choose between right and wrong—which, for practical purposes, is equivalent to not knowing the difference between right and wrong?

The author further states that the controversy between psychiatry and jurisprudence results from the fact

> that the alienist, first of all tries to form a general conception of the patient's conduct which is considered an unbroken continuity. For this medical expert then, the incriminated act of the insane offender is incidental to a more complex phenomenon of the history of the disease. The jurist on the contrary, is primarily concerned with the fact of the offense itself and is often inclined to treat the sum total of the defendant's mental characteristics as a mere incident to the general picture of the criminal event.

Since Mr. Brasol claims to ally himself with the more progressive elements and in particular since he claims to be a "scientist," he may be informed that it is the old formal and legalistic school of criminology that treated crime apart from the personality of the criminal, and that the more advanced view is that crime is but a symptom of diseased personality at the psychological level and that it cannot be understood without the personality of which it is but a partial expression. The difficulty lies in the fact that Mr. Brasol wholly fails to appreciate the psychiatric point of view, already permeating many court rooms, but still maintains views of hun-

dred and more years ago. To him psychiatry and jurisprudence are at the opposite poles and in spite of his presumed understanding attitude towards psychiatry, he has not been able to effect a synthesis between the two disciplines. On another occasion,* the reviewer, discussing the relation between psychiatry and jurisprudence, pointed out that although considerable differences exist between medical science and jurisprudence, they are by no means irreconcilable and that a more tolerant understanding between the two is quite possible. Through the influence of psychopathology, the law is coming to accept the pathologic significance of many sexual crimes as well as the factor of irresponsibility in the instance of grosser forms of mental disease. But much enlightenment is to be done yet, for the law is not yet ready to recognize the more molecular forms of mental disease, as well as the fact that mental diseases are primarily emotional disturbances, and therefore an individual with a clear intelligence may none the less suffer from a serious mental disease.

Since Mr. Brasol apparently fails to understand the meaning of treating crime apart from the criminal and of punishment as a corrective influence, it may be worth while to digress and discuss some of the essential aspects of crime and punishment as viewed by psychiatry.

The history of the human family does not record any instances of a society in function that has not had its criminal element. That the anti-social reaction should find so universal an expression, irrespective of the form or stage of society, suggests that the roots of criminality go beyond the particular social conditions under which it happens to express itself and that it flows out of some fundamental human tendencies common to all men at all times.

In dealing with the problem of crime, society has been guided by certain assumptions or premises the correctness of which were never adequately sustained. The first premise predicates that crime is a deed wilfully committed for personal motives of gain, material or otherwise. The second premise predicates that punishment is a deterrent of crime in that it serves as both a lesson for the transgressor and a warning to the rest of as yet innocent citizenry; and that its beneficial influence is in proportion to its

* The psychoanalytic Review Vol. XIII, January, 1926, Chapter VI.

severity. The fact that no system of laws or method of punishment, nor any form of education has yet been devised that can adequately cope with the problem, strongly suggests that neither of these assumptions is quite tenable. It is justifiable therefore to question whether, to begin with, crime is a wilful deed, whether gain is its main motive, and further, whether punishment is the deterrent that it is supposed to be.

In the recognition that in isolated instances at least, crime appears to have been committed under the stress of an uncontrollable drive and thus seemed to lack the element of wilfulness, we have the fruitful suggestion that crimes may be unconsciously motivated which makes punishment in such cases useless since the commission of the crimes is not within the control of the individual. Although it was believed that originally such acts are committed on impulse and only at the height of some great mental or psychic disturbance (epilepsy, paresis) which precludes the use of normal faculties, it is now being recognized that a mental or psychic abnormality may be continuous in operation no less than sudden and explosive, that an individual may not show obvious or gross abnormalities yet be abormal, may be perfectly composed, calmly deliberating and planning crime, yet fundamentally be driven by an unconscious urge and therefore not responsible for the act (paranoia). The net implication of this is that although a criminal may have a conscious motive, it does not preclude his having an unconscious motivation as well; and it is the latter that is the driving force. Since psychopathology has already made the contribution that in every act of human behavior there is, besides the conscious motive, an unconscious motive as well, and minute mental abnormalities are no less significant than the gross deviations, we may extend the principle to all criminal deeds and thus submit that every criminal act has a conscious as well as an unconscious motive which thus puts the entire problem of criminology within the scope of psychopathology. It will then be seen that the original premise spoken of, namely, that crime is a wilful deed is not correct and that although apparently wilful, the act is really unconscious and that behind the apparent motive of gain there is yet a far deeper motive. If this be granted then the second premise, that of punishment, also loses its foundation for we have already recognized that if a deed is unconsciously motivated, punishment can have no de-

terrent effect. Herein perhaps lies the reason for the failure of laws and punishment to cope with crime; because in the last analysis and in every analysis, crime is an unconsciously motivated reaction.

For centuries the treatment accorded the criminal was with regard to the act of crime and without reference to the individual perpetrating it and the circumstances surrounding it. In extent the punishment was quite out of proportion to any damage the act may have occasioned, an attitude well expressed in the Draconian law which in certain ages was a rather customary expedient in many communities. The attitude was no doubt charged with a great deal of emotion, the criminal apparently serving the purpose of a buffer or " shock absorber " for the undischarged antipathic and criminal trends of the citizen; the so-called " scapegoat." * A more reasonable attitude was assumed when the punishment meted out was made to depend on the nature of the crime and the degree of possible damage sustained by the injured party and the community; and still greater progress was made when further regard was given to the circumstances under which the crime was committed. More recently we have even come to inquire into the mental reaction of the individual criminal, and if such is found to be grossly abnormal he is relieved from the responsibility for the crime. The next step, already taken in some institutions. is to view crime as a symptom of some emotional disturbance, to study the personality of the criminal as a whole and with the knowledge of the psychic factors gained to so redirect his life as to make him better adjusted to himself and to the community; for he who is well adjusted has no need for transgression.

As Mr. Brasol is so very fond of quoting Russian authors, Dostoevsky in particular, the reviewer is tempted to match Mr. Brasol and also quote from Dostoyevsky. In " Buried Alive " Dostoyevsky gives a vivid description of the inside life of a prison and in the description makes the following pertinent comments on the psychology of crime:

It might perhaps seem hardly probable that in all those years I have never met with any signs of shame and contrition on the part of the convict, with

* For an admirable discussion of the psychic mechanisms in punishment see White, Wm. A., " Insanity and Criminal Law," Chapters II, III, *et seq.* For the discussion of the " scapegoat " see the same author " Principles of Mental Hygiene," Chapter V.

one instance, at least, of moral suffering caused by the memory of some crime; on the contrary, they seemed actually to pride themselves on being convicts and being sentenced to penal servitude, as if the very name of convict were an honorable appellation conferred only on a chosen few; . . . . I have frequently heard convicts relate the most terrible crimes, the most unnatural deeds, laughing heartily at the recollection of them. . . . . I suppose that this apparent hardness of heart is in great deal caused by false shame and bad example. On the other hand, who can say that he has seen into the inmost depths of those hearts and read there what is hidden from all the world?

The philosophy of crime is more difficult than is commonly supposed, and it is impossible to define crime according to certain points of view. Neither imprisonment nor the hard labor system will ever make the prisoner a better man or a useful member of society; while they are the means of punishing him and protecting society against his violence, they only develop in him a feeling of intense hatred, a thirst for forbidden pleasures, and an almost incredible recklessness. I am also persuaded that the solitary imprisonment system, in spite of all that has been said in its favor, entirely fails to fulfill its objects. It takes all the strength out of a man, enervates and weakens him morally, and terrifies him into becoming what is commonly called a model of repentance, but what really is no more like true repentance than a mummy is like a living, breathing human being.

No convict looks upon hard labor as an occupation. To him it is merely the hateful task which must be done, and as soon as he has finished it, or worked the stated number of hours, he goes back to the prison, where he devotes most of his spare time to some more profitable occupation than working for the Government. No man can exist without work, and the convict prisoner least of all.

Neither did the work in itself appear to me very difficult, and it was not till later that I began to realize that it was rendered irksome and almost unbearable through being imposed as a task which had to be finished by a certain time for fear of punishment. Many a poor laborer who is free works perhaps harder than a convict, and even spends sometimes a part of the night working out-of-doors, especially in the summertime. But he works for himself only, and this thought, and the knowledge that he will profit by his labor, is enough to reward him, while the convict is obliged to work at something which can never be thought of the slightest use to him. I have sometimes thought that the way to crush and annihilate a human being completely would be to set him to do a completely senseless and useless thing. Now, although the work executed by the convict is unprofitable and tedious so far as he himself is concerned, it is far from being aimless in itself. He makes bricks, works in the fields, builds houses, etc. Sometimes he even gets interested in his work, and tried to do it better and quicker than his fellow-workmen. But if he were condemned to pour water from one tub to another, and then back again, or to pound sand into a mortar, or to carry a heap of earth backwards and forwards, I am convinced that he would either commit suicide within a few days or murder some of his fellow-sufferers in order to suffer death at once and be delivered from this moral torture, shame, and degradation.

The whole job seemed to be merely a pretext to keep us busy. This the men knew, and went about their work in a listless manner, very different from the eagerness with which they worked at a job that offered more interest, especially if it was to be finished by a certain time. In such cases they seemed to grow quite excited over their task, and often would exert themselves to their utmost to finish it in a shorter time than had been allotted to them, not for the sake of the profit, for profit there was none for them, but because their self-love was touched.

All people do not hope alike—*e. g.*, a prisoner in a convict prison and a free man. The latter has always a distinct object in view, in hoping, *e. g.*, a change in his outward circumstances or the fulfillment of some desire; but at the same time his mind and body may be actively employed, and the very cares of life often prevent him from becoming too much absorbed in his hopes. The prisoner, it is true, is also actively employed; but he works and lives in a prison, and whatever his sin may have been, and however well deserved his punishment is, he instinctively demurs against accepting his sentence as his final destiny. The convict persists in looking upon his cell as a temporary abode where he is not, cannot be, at home. Twenty years of servitude dwindle into a mere nothing in his eyes, and he fully believes that he will leave the prison at fifty-five feeling as young and strong as he does at thirty-five.

For they know full well that they must spend the rest of their lies in chains and in prison till death sets them free. Yet they count the days when they will no longer be chained to the wall, for if this punishment were to be endless, could they bear it without dying of despair or going mad?

Love of money is one of the characteristic traits of the convict prisoner. He prizes money beyond everything next to liberty; and is never happier than when he can rattle it in his pocket. Without it he grows sad, restless, low-spirited, and will even do something desperate—rob his comrades, or do something worse—rather than be penniless. Yet, although money was such a valuable and much-valued object in the convict prison, it was a difficult one to keep. And they were all terrible thieves. We were allowed to have boxes with a lock and key to keep things in, but this did not prevent the men from helping themselves to their fellow-prisoner's property.

I was forcibly struck on this first day of my new life by the curious fact that all those who are not convicts and only come into daily contact with them, beginning with the sentinel at our gates and ending with the governor, should have the most exagerated ideas about convict prisoners. They seem to be constantly haunted by the fear that the convicts are on the point of committing some horrible crime, some desperate deed, and take vengeance on the unoffensive causes of their terror by bullying them and brow-beating them without the slightest provocation on their side. The prisoners are not ignorant of the terror they inspire, and are rather proud of having such a reputation. Yet they love and respect their superiors best who are not afraid of them and show by their behavior that they trust them.

I do not wonder that people who have never come into contact with prisoners, and in whose minds the words "convict prisoner" are associated with horrible tales of brutal crimes, cruelty, incendiarism, highway robbery, should experience a certain sensation of fear and disgust at the sight of a group of men accoutered in the grotesque prison garb, with half-shaven heads and marked on the brow and cheeks with the signs of their shame; but there is a good deal of exaggeration of their fear. A convict, even if he should be the most daring of men, does not pounce upon a fellow-prisoner and stab him without any provocation, except in very rare cases, when the deed is committed not from any personal motive of revenge and hatred, but merely of saving himself from iminent punishment by being brought up for trial for a fresh crime.

I have also wondered at the total absence of all feeling of ill-will with which these poor fellows used to talk about their punishment and those who punished them. Tales of cruelty which would make my heart throb with indignation were told by the sufferers without the slightest shade of anger or hatred; not infrequently they would laugh like children while relating their sufferings.

These are gems of psychologic insight which it would do well for Mr. Brasol to ponder over. Through years of confinement Dostoyevsky has had ample opportunity to observe the intimacies of prison life. His one unequivocal conclusion is that imprisonment and punishment are not deterrents of crime, but on the contrary provocations and stimuli for further crimes. Back of that is the realization that the crime is not a wilful act which explains, in part at least, the reason for the failure of punishment. Perhaps if the criminal were not punished, the inner remorse which no doubt overtakes many, would cause him far greater suffering than any punishment that the law could mete out. But Mr. Brasol would insist that crime is a wilful deed, and that punishment is the proper deterrent of crime. And this is supposed to be an enlightened scientific view! What a travesty on science and enlightenment!

To sum up: Mr. Brasol's book is a worthless contribution to a problem that deserves much better treatment than this. It is not a work based on mature experience and careful observation, but entirely "made to order" to defend a prejudice and a preconception. It is full of faulty logic, immature, even puerile thinking, gross inaccuracies and misinterpretations. Although a large literature is quoted, it was obviously read to find facts to support an argument rather than evaluate the data in the light of the new knowledge gained. The reviewer has gone to the trouble of dissecting the book at length, not because he thought there was any

inherent value in the book itself; but because there is a danger that might be accepted as authoritative. There is much more in the book to criticize than appears above, indeed almost every statement is a challenge to common sense. Lest he might be accused of having taken the meaning out of its context, the reviewer has quoted the author at length and in his own words. In the reviewer's opinion, this is a dangerous book because of its total lack of critique, of absence of scientific approach, and therefore a book that is likely to impress the unsuspecting reader with the same errors and prejudices with which the author is filled. In all, Mr. Brasol has failed to accomplish the task he set forth to accomplish. His interpretation is "scholastic, formal, legalistic and based on abstract formulas." He has failed to offer us an explanation of crime in terms of biology, psychology, sociology, anthropology and economics, but instead gave us a jumbled and unassimilated mixture of these disciplines. And the criminal still remains an enigma.

Notes and Comment.

Dr. C. M. Hincks Appointed General Director National Committee for Mental Hygiene.—On January 1, 1931, Dr. C. M. Hincks of Toronto, Canada, was appointed General Director of the National Committee for Mental Hygiene. Since its organization in 1918, Dr. Hincks has been identified with The Canadian National Committee for Mental Hygiene. During the six-year period between 1918 and 1924, he was associated with Dr. C. K. Clarke who was the first Medical Director of the Canadian organization. Following the death of Dr. Clarke in 1924, Dr. Hincks became Medical Director.

The Canadian National Committee for Mental Hygiene has been instrumental in securing the active co-operation of the Federal and Provincial Governments in the promotion of mental health progress. Provincial mental hygiene surveys conducted by the committee have led to the improvement of therapeutic facilities for the mentally ill and to the expenditure on capital account of approximately twenty million dollars. A notable feature of the committee's work has been the promotion of mental hygiene research at four Canadian universities.

Dr. Hincks will retain his connection with the Canadian Committee as Director. He will be assisted by Dr. Grant Fleming, Professor of Preventive Medicine and Public Health, McGill University, who becomes Medical Director, and by Dr. C. B. Farrar, Professor of Psychiatry, University of Toronto, who has joined the staff as Associate Medical Director.

It will be Dr. Hinck's aim to co-ordinate the activities of the National Committees of both countries and to work in the closest possible relation with The American Psychiatric Association. He believes that National Committees can do much to promote basic mental hygiene research and to give impetus to the application of mental hygiene principles in education, medical practice, nursing, social work industry, religion and in the administration of law. He is of the opinion that progress will come about through secur-

ing the intelligent interest of the public in mental hygiene matters and through the giving of the people at large a sense of partnership with scientific leaders.

In the preventive phases of a mental program, Dr. Hincks desires a large measure of leadership from psychiatrists, but in addition he considers that the whole-hearted participation of educators, psychologists and sociologists is necessary. Of immediate urgency, in his opinion, is the re-casting of medical education so that the rank and file of graduates from medical schools will have an appreciation of personality and environmental factors in the general run of medical practice.

Since Dr. Hincks spent six years in survey work, he is particularly interested in mental hospital organization and in the various facilities for the treatment and control of mental disabilities. He hopes that satisfactory arrangements may be made to raise the scientific standards of mental institutions and to promote the organization of extra-institutional activities.

Dr. Hincks was born April 8, 1885, at St. Mary's, Ontario, of English parentage. His father, Rev. Dr. W. H. Hincks, was for fifty years pastor of various Methodist churches in various parts of Ontario, but mostly in and around Toronto.

He was graduated in Arts from the University of Toronto in 1905, and in Medicine in 1907. Upon graduation he engaged in general practice in Toronto, continuing until his appointment as Medical Inspector of Schools for Toronto in 1913. This work brought him in touch with mentally retarded children and his interest quickly advanced to concern for mental maladjustment at the adult level, and led to his becoming one of the founders of the first mental hygiene out-patient clinic in Canada.

This wider contact with mental maladjustment led in 1918 to his organizing The Canadian National Committee for Mental Hygiene, of which he was at first Secretary and later (1924) Medical Director. It can be said that a very large share of whatever advance has been made in Canada in the direction of mental hygiene, appreciation and application is due to his efforts and to those whom he has in one way or another from time to time induced to co-operate with him.

TRIBUTE TO DR. FRANKWOOD E. WILLIAMS.—At the twenty-first anniversary meeting of the National Committee for Mental Hygiene, held in New York in November last, Dr. William A. White, referring to the prospective retirement of Dr. Williams on January 1, 1931, from the directorship of the National Committee, said that Dr. Williams had at the request of the Executive Committee held in abeyance for two years the resignation which had recently been accepted. After referring to Dr. Williams' fourteen years service with the National Committee, first as Associate Medical Director, and then as Medical Director, Dr. White said:

> His term of office was coincident with a period of the organization's most active growth. I like to think at this moment of the National Committee as having passed through its early youth under the directorship of our late lamented Dr. Salmon, and as having passed through its adolescent period and having come of age under Dr. Williams's directorship. Dr. Williams had the very difficult task of substituting for the dreams and fantasies of the child, so to speak, the more definite goals and realities which were to serve as objectives for its future progress as it merged into adulthood, and those of us who have been closely associated with the mental hygiene movement know the wisdom of his guidance. We know how he has touched this movement at many of its most vital points. To have maintained the leadership, as he did, of a scientifically directed group, is an achievement of the greatest significance.
>
> As a teacher and writer, as advisor and consultant, Dr. Williams exerted a wide influence, shaping the thought of all workers in mental hygiene and related fields. To his leadership, energy and vision are due the stimulation of mental hygiene work in the colleges and universities, the training of psychiatrists and psychiatric social workers for extra-mural mental hygiene work, the sound professional orientation of the child guidance movement, and the infiltration of mental hygiene through medicine, education, social work, criminology and other fields of effort. He formulated the program of the First International Congress on Mental Hygiene, held in Washington, last May, and was responsible, to a very considerable degree, for the success of that great conference.

DR. ADOLF MEYER TO BE THE FIRST THOMAS W. SALMON LECTURER.—A meeting in memory of Dr. Thomas W. Salmon was held at the New York Academy of Medicine, on Saturday evening, January 10, 1931.

At this meeting the completion of the fund of $100,000.00 which was raised by friends and admirers of Dr. Salmon was announced.

The custody of this fund has been vested in the New York Academy of Medicine and the lectures will be delivered under its auspices and the lecturer selected annually by the Academy and a special advisory committee.

Under the terms governing the trust, a scientific worker who has made outstanding contributions to psychiatry, mental hygiene or a related field, must be chosen to deliver the lecture or lectures.

The academy and its advisory committee have, in the most wise selection of Professor Adolf Meyer as the first lecturer, honored American psychiatry and made an excellent choice.

We predict that Professor Meyer will establish a standard in these first lectures which it will be most difficult for his successors to attain.

We congratulate the Academy upon its selection and Professor Meyer upon the opportunity before him as well as upon the honor conferred upon him.

Association and Hospital Notes and News.

American Psychiatric Association, Arrangements for Round Table Conferences.—The Committee on Program has issued the following notices in reference to the forthcoming annual meeting of the Association in Toronto from June 1 to 5, 1931:

The Committee on Program is prepared to give every assistance to groups who wish to discuss some problem informally. To this end, a Round Table will be provided on the request of three or more members. The committee will not arrange programs for these groups but will arrange for a meeting place, provide a chairman if one is not beforehand designated by the group, and inform the Association of the opportunity for others to sit at the table. It may be remarked that two new groups are in prospect already, and there will probably be tables to discuss "The Problems of the Private Institution," and "Psychoanalysis in Hospitals."

Census of Hospital Activities, Staff Vacancies, Etc.—The blank questionnaire sent out several months ago still remains unfortunately, in several instances unanswered. The blanks had a wide circulation, and have in many instances been returned quite satisfactorily filled out.

This work was undertaken for the benefit of the hospitals for mental disorders throughout the country. If a sufficient number of the blanks is returned to afford a basis for certain conclusions from the data thus obtained, we believe that our hope of accomplishing something of value for the Association and for the hospital will be fulfilled.

The larger the number of returns we have the smaller the probability of error in any deduction which may be drawn therefrom. We therefore urge upon all who have thus far neglected to answer the questionnaire to do so at once and return the filled out paper to us.

It is highly desirable that the compilation from the returns which have been received or which may be sent in following this appeal

be completed some weeks before the annual meeting that certain committees may take advantage of the data thus obtained in preparing their reports.

APPLICANTS FOR FELLOWSHIP OR MEMBERSHIP IN THE AMERICAN PSYCHIATRIC ASSOCIATION.—The following list is published in conformity with the rules of the Association. A supplemental list will appear in our March issue:

FOR FELLOWSHIP.

Benjamin W. Black, M. S., Piedmont, Calif.
C. P. Kjaerbye, M. D., Fresno, Calif.
Chester A. Marsh, M. D., Selinsgrove, Pa.
Rose D. Senior, M. D., Chicago, Ill.
Edward W. Twitchell, M. D., San Francisco, Calif.

FOR MEMBERSHIP.

Robert G. Armour, M. D., Toronto, Ont., Canada.
Prince P. Barker, M. D., Tuskegee, Ala.
Frank Berner, M. D., New York, N. Y.
George F. Boyer, M. D., Toronto, Ont., Canada.
George C. Branche, M. D., Tuskegee, Ala.
Isra T. Broadwin, M. D., New York, N. Y.
Maudie M. Burns, M. D., Poughkeepsie, N. Y.
Ernest A. Clark, M. D., Toronto, Ont., Canada.
Robert Cumming, M. D., White Plains, N. Y.
James A. Cutting, M. D., Agnew, Calif.
William H. Dunn, M. D., White Plains, N. Y.
Edwin R. Eisler, M. D., Chicago, Ill.
Frank G. Engler, M. D., Wichita Falls, Texas.
Bernard S. French, M. D., Watkins Glen, N. Y.
Arnold Gesell, M. D., New Haven, Conn.
Louis P. Harshman, M. D., Fort Wayne, Ind.
William C. Herriman, M. D., Cobourg, Ont., Canada.
James Houloose, M. D., Long Beach, Calif.
Goldun W. Howland, M. D., Toronto, Ont., Canada.
Simon O. Johnson, M. D., Tuskegee, Ala.
Archibald J. Kilgour, M. D., Kingston, Ont., Canada.
Allie C. Kolb, M. D., Louisville, Ky.
Arthur E. Landers, M. D., Reno, Nevada.
Clarence H. Lewis, M. D., Kingston, Ont., Canada.
Alexander S. Lorand, M. D., New York, N. Y.
Leslie E. Luehrs, M. D., New York, N. Y.
Harry E. Marselus, M. D., East Moline, Ill.
Honora McCarty, M. D., Agnew, Calif.
Gordon A. McLarty, M. D., Toronto, Ont., Canada.
Walter G. Niles, M. D., Chattahoochee, Fla.
Robert C. Montgomery, M. D., Whitby, Ont., Canada.
Harold D. Palmer, M. D., Philadelphia, Pa.
Hyman L. Rachlin, M. D., New York, N. Y.
William E. Render, M. D., Louisville, Ky.
Karl Rothschild, M. D., New Brunswick, N. J.
David Rothschild, M. D., Foxborough, Mass.
Charles D. Ryan, M. D., White Plains, N. Y.
Hervey B. Scott, M. D., Louisville, Ky.
John N. Senn, M. D., Hamilton, Ont., Canada.
Dudley D. Shoenfeld, M. D., New York, N. Y.
Glenn J. Smith, M. D., Jackson, La.
Alfred P. Solomon, M. D., Chicago, Ill.
Franklin C. Southworth, M. D., Framingham, Mass.
Hamlin A. Starks, M. D., White Plains, N. Y.
Miguel Steinberg, M. D., New York, N. Y.

AMERICAN ORTHOPSYCHIATRIC ASSOCIATION.—The eighth annual meeting of this Association will be held at the Hotel Pennsylvania, New York, on February 20-21, 1931.

This Association has established The American Journal of Orthopsychiatry. A Journal of Human Behavior, which will be the official organ of the Association. It is edited by Dr. Lawson G. Lowrey and a group of associates and issued quarterly.

Two numbers have been issued and present an attractive appearance and a varied, well-selected and interesting table of contents.

AMERICAN OCCUPATIONAL THERAPY ASSOCIATION.—At the half-yearly meeting of the Board of Management of the American Occupational Therapy Association, held on January 3, 1931, in Philadelphia, Pa., with the President, Dr. Joseph C. Doane, of Philadelphia, in the chair, Dr. Frank Billings, of Chicago, Ill., who was chief of the Reconstruction Division of the office of the Surgeon-General of the U. S. Army during the war period, and Dr. Goldwyn W. Howland, of Toronto, Canada, were elected honorary life members of the American Association in recognition of their valuable services in the development of modern methods in, and the wider use of, curative occupations for the sick and the disabled.

INTERNATIONAL NEUROLOGICAL CONGRESS.—The preparations for the International Neurological Congress to be held in Berne, August 30 to September 4, 1931, have continued to progress during the past months.

The local Swiss committee has been preparing plans for suitable entertainments. Tentative plans have been made for receptions, evening entertainments in the vicinity of Berne and for a day's trip to Interlaken, the Lakes of Thun and Brienz and the vicinity of the Jungfrau. Definite announcements in regard to the entertainment will be forthcoming in the near future.

The sessions of the Congress will be held in the Municipal Casino which will afford ample accommodations for the delegates, members of their families and guests. The large gallery will be reserved for members of the family and guests who may be enrolled at the time of the Congress as affiliated members at a moderate fee. Only members of the Congress will be admitted to the floor of the

Casino. Neurologists and psychiatrists in the United States, members of psychiatric societies or those whose chief interest is in these two fields are eligible to membership in the Congress. Application forms for membership may be obtained from the secretary-general of the Congress, Dr. Henry Alsop Riley, 117 East 72d Street, New York, N. Y.

The firm of Thomas Cook and Son has been appointed as the official travel agency and has prepared a very attractive itinerary, one trip being to the north and east and the other to the south and west, both including Paris and ending at Berne two days before the opening of the Congress. The financial terms for these trips of six weeks, in which are included all necessary expenses, transportation, hotel accommodations, etc., are very moderate. Detailed information in regard to these trips can be obtained from Thomas Cook and Son. On account of the fact that the tentative reservations for accommodations on the French Steamship Liner de Grasse and for the hotels on the proposed tour cannot be held beyond a date early in 1931 unless definitely reserved, it is necessary for those planning to take either one of the tours to make at least tentative arrangements with Thomas Cook and Son at once. Individuals wishing to have special itineraries can do so by arrangement with Thomas Cook and Son. From present indications the number of American neurologists and psychiatrists with their families will require almost one-half of the total accommodations available in Berne, so that if arrangements are not made through Thomas Cook and Son at an early date, many members of the Congress will have to be satisfied with inferior arrangements or reservations outside of Berne. Thomas Cook and Son have complete control of the available accommodations in Berne and it will be well nigh impossible for individuals to obtain proper accommodations unless they secure them from Thomas Cook and Son.

Neurologists and psychiatrists intending to attend the Congress and wishing to present papers before it, were urged to send to the secretary-general titles and abstracts not later than December first. It will be impossible to consider titles and abstracts submitted after that date.

According to preliminary calculations a very large participation of neurologists and psychiatrists from all parts of the world is

already assured and the Congress should be a milestone in the history of neurology and psychiatry.

The following preliminary programs have been arranged for the various subjects which will be presented during the morning sessions (and one afternoon session) of the Congress:

1. Diagnostic and Therapeutic Procedures (Surgical and Otherwise) in Brain Tumors.
   Program arranged by Prof. Max Nonne, Hamburg.
   Morning and Afternoon Sessions, Monday, August 31.

DIAGNOSIS.

I. Clinical Symptomatology.
   Reporter. Sir James Purves-Stewart, London.
   Co-reporters: Clovis Vincent, Paris, Giuseppe Ayala, Rome, Foster Kennedy, New York, Tumors of temporosphenoidal lobes—Subfrontal syndromes.

II. Histological Diagnosis.
   Reporter: Percival Bailey, Chicago.
   Co-reporters: Wilder Penfield, Montreal, Bertold Pfeifer, Nietleben, Germany, Callosal puncture.

III. Roentgenological Diagnosis.
   Reporter: Arthur Schüller, Vienna.
   Co-reporters: Ernest Sachs, St. Louis, H. W. Stenvers, Utrecht, Roentgenology in neuro-surgery.

IV. Ventriculography and Encephalography.
   Reporters: Walter Dandy, Baltimore, Tumors of the hemispheres and of the posterior cranial fossa; Otto Foerster, Breslau, Brain tumors—Pseudotumors.
   Co-reporters: Francis Grant, Philadelphia, Egas Moniz, Lisbon, Arterial iodide injection.

V. Sero-diagnosis.
   Reporter: Victor Kafka, Hamburg.
   Co-reporters: Frank Fremont-Smith, Boston, H. Bohnenkamp, Würzburg, Germany, Electrical resistance.

THERAPY.

I. Surgical Therapy.
   Reporter: Harvey Cushing, Boston.
   Co-reporters: Thierry de Martel, Paris; Otto Foerster, Breslau; H. Olivecrona, Stockholm; Charles H. Frazier, Philadelphia; Ludwig Puusepp, Tartu, Esthonia.

II. Roentgenological Therapy.
   Reporter: A. Beclère, Paris.
   Co-reporters: H. Cairns, London, Hirsch, Vienna, Radium.

III. Organotherapy.
Reporter: Tracy J. Putnam, Boston.

2. Muscle Tonus, Anatomy, Physiology and Pathology.
Program arranged by Sir Charles Sherrington.
Morning Session, Tuesday, September 1.

I. Anatomy.
1. S. Walter Ranson, Chicago, The nuclei and tracts concerned in the postural responses elicited by stimulation of the mesencephalic tegmentum.
2. Ken Kuré, Tokyo. On the tonus tracts and their terminal plates in muscle.

II. Experimental Physiology.
1. G. G. J. Rademaker, Leyden.
2. Graham Brown, Cardiff.
3. E. A. Spiegel, Vienna, Zur Pharmakologie der zentralen Tonusregulation.
4. L. Asher, Berne, influence of sympathetic nervous system on skeletal muscle.
5. Denny Brown, London. The part played by afferent muscular nerve-endings in postural (tonic) reflexes.
6. V. von Weizsäcker, Heidelberg, Electrical analysis of tonus.

III. Pathology.
1. F. Bremer, Brussels, Pharmacologie du tonus normal et pathologique.
2. Lewis J. Pollock and Loyal Davis, Chicago, The relation of modifications of muscle tonus to interruption of certain anatomical pathways.

IV. Clinical.
1. S. A. Kinnier Wilson, London, Disorders of tonus at different physiological levels, with special reference to the cortex.
2. R. Cruchet, Bordeaux, Relation of tonus to the Parkinsonian syndromes.
3. J. Ramsay Hunt, New York, The static and kinetic systems and their relation to muscle tone.
4. F. Negro, Turin, Alterations of muscle tonus in extrapyramidal syndromes.
5. A. Thévenard, Paris, Le phénomène de la poussée.
6. Concluding remarks by Sir Charles Sherrington.

3. Acute Non-Suppurative Infections of the Nervous System.
Program arranged by Prof. Georges Guillain, Paris.
Morning Session—Thursday, September 3.
Reporters:
1. Otto Marburg, Vienna, Introduction to the general pathology of acute infections of the nervous system.
2. Georges Marinesco, Bucarest, Pathogénie de certaines encéphalomyélites à ultra-virus.

3. J. G. Greenfield, London, The general pathologic anatomy of acute infections of the nervous system.
4. August Wimmer, Copenhagen, General clinical studies of acute infections of the central nervous system.
5. H. Pette, Germany, Rabies-Borna's disease. Landry's paralysis.
6. André Thomas, Paris, Zona—the Herpetic Infections.
7. L. Van Bogaert, Antwerp, Vaccinal encephalitis, vari-cellular encephalitis. Non-classified cases.
8. V. M. Buscaino, Catania, Psychiatric and biological questions in connection with acute infections of the nervous system.
9. Concluding remarks by Prof. George Guillain.

4. The Rôle of Trauma in the Production of Nervous Symptoms.
Program arranged by Prof. O. Rossi, Pavia.
Morning Session, Friday, September 4.

1. Professor Rossi, Importance and limitations of the subject. General discussion.
2. Charles P. Symonds, London, Commotio cerebri—diagnosis and treatment of the cerebral states following head injuries (exclusive of the ordinary brain injuries).
3. Jean Lhermitte, Paris, Spinal cord injuries (Commotio medullae spinalis.).
4. Arthur von Sarbó, Budapest, Injuries of the peripheral nerves. (Microscopic traumatic changes in the nervous system.)
5. O. Veraguth, Zürich, Rôle of trauma in producing some nervous diseases (cerebral tumor, amyotrophic lateral sclerosis, multiple sclerosis, etc.).
6. P. Rio del Hortega, Spain, Traumatic changes in the glia as explanations of some disorders of nervous function.
7. F. Naville, Switzerland, Effects of some special traumata (Electrocution, etc.).
8. Smith Ely Jelliffe, New York, The psychological level in trauma of the nervous system.

The arrangement of the programs for the afternoon sessions will probably be completed during the early part of 1931 and definite announcement will be made as soon as possible. The present plans of the Program Executive Committee are to hold sessions of the entire Congress on each afternoon. If however, the number of papers deserving to be presented is very large, it may be desirable to divide the topics under various headings and to hold separate sessions of the Congress on the various afternoons. The members of the Congress may then attend any particular session in which they have special interest.

Special clinics will be arranged for the members of the American delegation who are expecting to make the tours which have been arranged by Thos. Cook and Son. These clinics will in all probability be held at Paris, under the direction of Professor Guillain or one of his associates; at Amsterdam, under the direction of Professor Brouwer or one of his associates; at Zurich by Professors Maier and Minkowski, and at Pavia by Professor Rossi or one of his associates.

To enable the Secretary-General to complete the authoritative list of members, all applications for membership should be in his hands by April 15.

Book Reviews.

Epilepsy. By WILLIAM G. LENNOX, M. D., and STANLEY COBB, M. D. (*Baltimore: Williams & Wilkins, 1928, pp. 197.*)

Treatment of Epilepsy. By FRITZ B. TALBOT, M. D. (*New York: The Macmillan Co., 1930, pp. 308.*)

Since the latter of these publications refers in particular to the earlier, it seems proper to review the two. In the monograph by Drs. Lennox and Cobb, we have the most thorough exposition of the conception of epilepsy with which the reviewer has had the good fortune to make contact. The work is meticulously thorough; its statements are illuminating; and its data elaborate.

"For the present discussion, we define it [epilepsy] as a syndrome characterized by the sudden appearance of paroxysms, of which convulsive movements or loss of consciousness or both, are a principal element." "We shall confine ourselves to the presentation of evidence which may throw light on the mechanism involved in seizures, and on the cause and treatment." There follows a section on neurological considerations, including the irritation, release, short circuit, and explosion theories. "At the present writing it is the authors' belief that epileptic seizures are neurological phenomena, usually motor, caused by some sudden change in the nerve cells. The etiological factors which might spring the trigger of this mechanism may be numerous and varied." There follows a section on the factors involved in convulsions, including pathology of the nervous system, which "give us no etiological point of departure"; and evidence from clinical examinations, including ventriculography. "Almost any lesion plus the unknown *X*, which we call functional instability, may result in epilepsy." Functional abnormalities of the nervous tissue next concern us. "The problem is not unique for convulsions. Individual variation in the manner of reacting to environment is a characteristic of living matter. In the presence of a given stimulus, only certain individuals will react with a convulsion. This is no more mysterious than the fact that only certain individuals are 'susceptible' to a given infection, or to cancer." Physico-chemical changes are discussed: oxygen consumption, ionic equilibrium, hydrogen-ion concentration, edema and permeability of cell membranes, blood supply to the nerve cells, and chemical constituents. There follows a section of evidence from the effect of drugs. After summarizing this data, the matter of psychogenic factors is touched on briefly.

Abnormalities outside the central nervous system, circulatory system, automatic nervous system, respiration, gastro-intestinal tract, basal metabolism, endocrine glands, the blood, spinal fluid, urine, bacteriological factors, and factors of toxicity, are presented. Protein metabolism, the matter of ferments, carbohydrate and fat metabolism, acid-base equilibrium, including the effects

of induced alkolosis and induced acidosis, are presented, *in extenso*. There follows a very valuable section entitled "General Conclusions," each sentence of which deserves quotation. In seven pages there is presented a conspectus of the best available information in this field. "The encouraging feature is that, although the exact mechanism of seizures is unknown, much can be done to prevent and, in the early stages, to relieve them. . . . . Finally, no one can doubt that continued study and search will bring a better understanding and a more rational and effective treatment of this distressing symptom." A bibliography of some 650 titles, authors' index and a subject index, complete the monograph.

The more recent of the two books under review, while referring to the earlier one as a clear outline of the present-day conception of epilepsy, none the less devotes some 46 pages to historic and etiologic considerations. "What few indubitable facts we have seem to indicate that the fundamental disorder of epilepsy is of such a character that it affects all parts of the body." Chapter IV discusses diagnosis; Chapter V, prognosis; Chapter VI—of eight and one-half pages—symptoms; and Chapter VII—one and one-third pages—prophylaxis. "To outline the prophylaxis of epilepsy is very difficult, ..."

In Chapter VIII, treatment is outlined. A diagram, provided to show an overlapping of therapy by physical hygiene, social and mental hygiene, diet, and drugs, is included. The largest area is awarded to diet and, in fact, the second and largest section of the book is a discussion of dietary treatment with particular reference to the use of the ketogenic diet. The first section concludes with a brief chapter each on education, on recreation, and on medication.

The second section of this book is made up of eight chapters: factors necessary for an understanding of dietary treatment; summary of theories of dietetic treatment of seizures. "About all that can be said, therefore, in conclusion is that the ketogenic diet appears to be the most effective form of the dietetic treatment of epilepsy"; clinical results of fasting; clinical results of the ketogenic diet; the practical use of the ketogenic diet; and detailed management of the diet, are outlined. Chapter XVIII is entitled "What to teach an epileptic patient"; the final chapter of one paragraph is entitled "Migraine." A reprint of the report forms evolved for their study for the Association for Research in Nervous and Mental Disease, is inserted; there is a bibliography of 306 titles, and an index.

Dr. Cobb is Bullard Professor of Neuro-Pathology in the Harvard Medical School; Dr. Lennox, Assistant in Medicine and Research Fellow in Neuro-Pathology; and Dr. Talbot, Clinical Professor of Pediatrics, in the same medical school.

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